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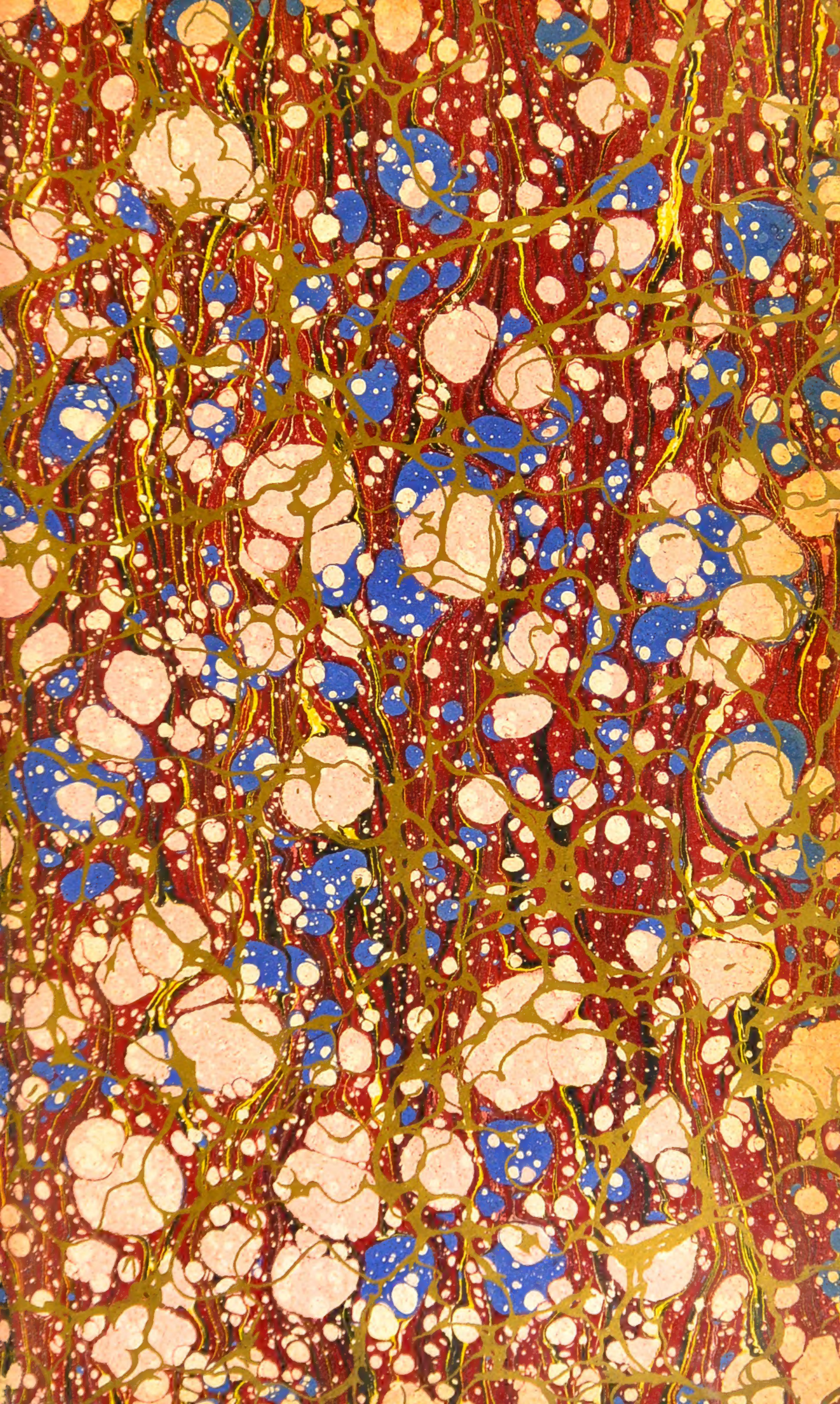

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TWENTIETH CENTURY PRACTICE

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BY

LEADING AUTHORITIES OF EUROPE AND AMERICA

EDITED BY
THOMAS L. STEDMAN, M.D.
NEW YORK CITY

IN TWENTY VOLUMES

VOLUME III.
OCCUPATION DISEASES, DRUG-HABITS, AND POISONS

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CONTENTS.

PAGE
ALCOHOLISM AND DRUG HABITS, ... 1
Definition of Terms, ... 4
Toxic Effect of Articles Taken to Excess, ... 6
Alcoholism, ... 6
Morphinism, ... 70
Cannabism, ... 88
Chloralism, ... 89
Chloroformism, ... 92
Etherism, ... 95
Cocainism, ... 99
Substances the Intoxicating Capacity of which is Doubtful, . . . 100
Gelsemium, ... 100
Sulphonal, ... 101
Paraldehyde, ... 101
Antipyrin, ... 103
Arsenicism, ... 104
Unintoxicating Substances, ... 106
Tobaccoism, ... 106
Theinism, ... 112
Caffeinism, ... 114
Narcomania (the Disease of Inebriety), ... 115
Relations of Inebriety to Insanity, ... 116
Periodical Inebriety, ... 119
Habitual or Constant Inebriety, ... 122
Social and Solitary Inebriety, ... 123
Etiology, ... 123
Pathology, ... 130
Treatment, ... 131
Medico-Legal Relations, ... 131
Relation of Inebriety to Insurance, ... 136
Bibliography, ... 137
SHOCK AND COLLAPSE, ... 139
Symptoms, ... 141
Causes, ... 144
Reaction from Shock, ... 155
Prognosis, ... 157
Pathology, ... 158
Diagnosis, ... 162
Prophylaxis, ... 163
Treatment, ... 164
Bibliography, ... 170

PAGE

SEASICKNESS, ... 173
  Symptoms, ... 176
  Causation, ... 181
  Nature, ... 188
  Treatment, ... 189
  Therapeutic Uses of a Sea Voyage, ... 195
  Bibliography, ... 196
MOUNTAIN SICKNESS, ... 199
  Symptoms, ... 202
  History, ... 210
  An Attempt to Explain the Symptoms, ... 215
  Summary, ... 225
  Acclimation, ... 226
  Diagnosis, ... 227
  Prognosis, ... 228
  Treatment, ... 228
  Bibliography, ... 229
OSTEOMALACIA, ... 233
  Deformities, ... 236
  Pathological Anatomy, ... 238
  Etiology, ... 240
  Pathology, ... 241
  Diagnosis, ... 246
  Treatment, ... 248
HEAT-STROKE, ... 251
  Definition, ... 254
  Symptoms, ... 258
  Causation, ... 272
  Morbid Anatomy, ... 275
  Prophylaxis, ... 276
  Treatment, ... 277
  Sequelæ, ... 283
  Bibliography, ... 285
FROST-BITE, ... 237
  Etiology, ... 291
  Symptoms, ... 294
  Morbid Anatomy, ... 297
  Treatment, ... 300
  Bibliography, ... 306
THE DISEASE OF OCCUPATIONS, ... 309
  Historical Note, ... 311
  Conditions of Occupations with Reference to the Prevalence of Disease, . 317
  Legal Restraints upon the Employment of Women and Children in the United States, ... 328
  Classification, ... 339
  Effects of Injurious Substances, ... 342
  Arsenic, ... 342
  Mercury, ... 348
  Lead, ... 355
  Phosphorus, ... 367

PAGE
Copper, . . . 373
Zinc, . . . 379
Tin, . . . 381
Chromium, . . . 382
Chlorine, . . . 384
Bromine, . . . 385
Iodine, . . . 385
Cyanogen Compounds, . . . 386
Sulphur and Its Oxides, . . . 386
Oxides of Carbon, . . . 389
Bisulphide of Carbon, . . . 391
Illuminating Gas, . . . 396
Aniline, . . . 397
Nitro-benzene, . . . 400
Turpentine, . . . 402
Alkalies, . . . 404
Quinine, . . . 405
Tea, . . . 406
Electricity, . . . 408
Dust, . . . 411
Anthrax Bacillus, . . . 428
Tobacco, . . . 431
Effects of Injurious Environment, . . . 433
Heat, . . . 433
Cold, . . . 436
Compressed Air, . . . 437
Rarefied Air, . . . 449
Concussion of the Air, . . . 451
Bad Sanitation and Ventilation, . . . 452
Effects of Injurious Exercise, . . . 457
Fatigue Neuroses, . . . 457
Laryngeal Affections of Professional Voice Users, . . . 458
Writer's Cramp, . . . 462
Telegrapher's Cramp, . . . 473
Miscellaneous Forms of Fatigue Neuroses, . . . 481
Miner's Nystagmus, . . . 484
Effects of the Sewing-machine, . . . 486
Neurasthenia from Overwork, . . . 488
Effects of Constrained Attitudes, etc., . . . 493
POISONING, . . . 497
Aconite, . . . 499
Belladonna, . . . 507
Calabar Bean, . . . 514
Cocaine, . . . 517
Cocculus Indicus, . . . 520
Colchicum, . . . 523
Curare, . . . 526
Digitalis, . . . 528
Gelsemium, . . . 533
Hellebore, . . . 535

PAGE
Hemlock, ... 536
Hyoscyamus, ... 539
Opium, ... 542
Solanum, ... 553
Stramonium, ... 555
Strychnine, ... 557
Veratrum, ... 566
Sulphuric Acid, ... 568
Nitric Acid, ... 570
Hydrochloric Acid, ... 571
Carbolic Acid, ... 573
Hydrocyanic Acid, ... 576
Oxalic Acid, ... 580
Soda and Potassa, ... 582
Ammonia, ... 583
Chloral Hydrate, ... 585
Illuminating Gas, ... 587
Lead, ... 593
Arsenic, ... 606
Zinc, ... 611
Copper, ... 611
Mercury, ... 613
Silver, ... 616
Phosphorus, ... 618
INDEX, ... 623

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ALCOHOLISM AND DRUG HABITS.

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ALCOHOLISM AND DRUG HABITS.

THERE is no form of poisoning which is so widely spread and so rapidly increasing at the present time, as the series of toxic phenomena exhibited in the persons of those addicted to the excessive use of the various preparations of alcohol, opium, morphine, chloral, chloroform, ether, cocaine, and allied inebriating substances. There is no other kind of poisoning which so degrades brain structure and disturbs mental function, while physically degenerating bodily texture and undermining vital organs, or which is so far-reaching in its operation, transmitting as it does through two generations a great variety of brain abnormalities, mental defects, and moral obliquities originally induced in the ancestral stock by the toxicating action of the poison on brain and nerve tissue.

In the consideration of the very wide subject of inebriate phenomena two great groups of morbid symptoms present themselves. One group consists of the toxicating effects produced on body and brain by the injurious influence of the poisonous articles on tissue, organ, and function. The other group comprises the diseased conditions which, in whole or part, operate in certain individuals to impel them to "take to drink," or to give themselves up to other forms of narcotic or anæsthetic indulgence.

The former group of disease manifestations includes those caused by two varieties of lethal drugs and other substances. The more generally employed of such articles, narcotics and anæsthetics, by their characteristic influence have the property of setting up and thus practically originating a desire for a fresh dose, and at the same time of so disturbing brain-function as to induce moral perversion. Such substances are alcohol, opium, morphine, cocaine, chloroform, and the like, the first-named—alcohol—being a prolific inciter to breaches of human and divine law ranging from minor offences to the gravest misdemeanors and crimes.

The second group of disease manifestations, which do not cause moral degradation and which incite to no deeds of violence or turpitude except in the rare cases in which insanity results from long-continued excess, comprises those arising from intemperance in such substances as tobacco, tea, coffee, and potassium bromide. The excessive use of such articles, as of various drugs which have been habitually and excessively indulged in (for on some constitutions any substance may make such an abiding impression as will stamp itself on the individual subject unless by a supreme effort it be grappled with), may cause considerable damage to the system; but, except in a sequela of mental unsoundness, material structure and bodily function are alone immediately affected, the *morale* and the conscience either remaining unimpaired, or being injuriously influenced only as a remote result of preceding physical pathological depravity. Excess in alcohol is daily credited, in police courts and before higher judicial tribunals, with offences against morality, law, and order; excess in coffee or tobacco never.

Till about a century ago, when Benjamin Rush laid down that habitual drunkards were diseased persons, with the exception of occasional utterances by a few thoughtful and far-seeing medical philosophers from the time of Hippocrates downward, there was a general belief that drunkenness was a purely voluntary condition, that men and women could get drunk or refrain from getting drunk as they chose, and that intoxication was a pastime willingly and wilfully indulged in; nor is this erroneous belief wholly dead. The many who formerly held and the comparatively few who still cherish this opinion, looking upon the whole matter of drunkenness as a mere immorality, vice, or crime, see no need for any medical consideration of the subject; punishment in a jail, confinement in the "stocks," pulpit denunciation, ecclesiastical excommunication, or the administration of a good whipping, being in their view the proper mode of dealing with such wanton and depraved outcasts. But modern scientific research has revealed the fact that a large proportion of the most confirmed topers are what they are though they have an utter loathing against intoxicants, and in spite of repeated and desperate struggles to avoid intoxication.

Medical science has, therefore, set itself to investigate thoroughly the nature and properties of intoxicant action on body and brain, and the various morbid conditions which operate to render a substantial proportion (happily but a minority of drinkers) of human beings an easy prey to some form of that enslavement by excessive indulgence in intoxicants which is the most complete possession and tyranny endured by men.

Definition of Terms.

It is imperative, at the outset, that there should be a clear comprehension of terms. So great have been the confusion and misunderstanding, often leading to apparent opposition of opinion when no opposition really existed, that in the most recent medical dictionary (Gould's) acute alcoholism is stated to be "inebriety;" and in most writings "inebriety" is employed as a synonym for "drunkenness." To remedy this loose nomenclature and to avoid the consequent general confusion of terms, expression, and thought, the writer has ventured to restrict the application of the term "inebriety" to that overpowering morbid impulse, crave, or craze, which tends to drive certain individuals to excess in intoxicants. To still further guard against the confusion of "inebriety" with "drunkenness," he has suggested an alternative scientific term to "inebriety" (inebriety the disease) in the word "narcomania," which is literally a torpor-mania (*νάρκη*, torpor)—in other words, a mania for narcotism by any intoxicating or anæsthetic substance. The crave is not for alcohol, or opium, or any like article, but for intoxication, or rather the temporary relief or satisfaction afforded by intoxication. The disease of inebriety (or narcomania) is thus a true intoxication-mania, the diseased inebriate often utterly abhorring the liquor which he would barter his salvation to procure. Nor is a drunken act the disease of inebriety, any more than is a mad and fatal assault the disease of insanity. Drunken and insane deeds of a drunkard or a lunatic are the symptoms or manifestations of certain diseased brain-states, which either of themselves or on the application of appropriate exciting influences are incited to impel to the committal of these deeds.

Contrary to usual parlance "alcoholism" and "alcoholomania" are not synonymous with "inebriety," but refer to only a variety of that disease. Thus not all inebriety is alcoholic. There is a great extent of non-alcoholic inebriety, especially among Eastern peoples, in opium, Indian hemp, and preparations of these and similar narcotics.

Alcoholism and alcoholomania need not be regarded as having strictly the same meaning. For the present review of the whole subject, the latter term may be considered as signifying that variety of the disease of inebriety or narcomania, which consists in a mania for intoxication by alcoholic beverages, just as morphinomania may be accepted as designating that variety of the disease of narcomania which consists in a mania for intoxication by morphine. Alcoholism and morphinism would be usefully applied to the morbid brain and body disorders produced by excessive indulgence in the respective poisons of alcohol and morphine. Alcoholomania and morphinomania may be induced by other causes than excess in alcohol and morphine, alcoholism and morphinism only by such use of these narcotic drugs. The study of the disease of inebriety or narcomania of course involves the study of alcoholism, though in this article the toxic phenomena are considered apart from the overpowering crave for intoxication.

I. TOXIC EFFECTS OF ARTICLES TAKEN TO EXCESS.

A. Intoxicants.

ALCOHOLISM.

By the term alcoholism is meant the various pathological conditions, processes, and symptoms caused by the intemperate consumption of alcoholic intoxicants.

Alcohol is an irritant narcotic paralysant which from its irritant and caustic action on the tissues, especially on those of the mouth, alimentary canal, and stomach, cannot be comfortably or safely drunk unless the virulence of its topical inflammatory action is sheathed by greater or less dilution with water. In this way, the action of alcohol is modified, ardent spirits such as rum, whiskey, brandy, and gin, which are, speaking broadly, about one-half water, more rapidly and powerfully injuring the texture and function of vital organs, like the liver and kidneys, than wine which is generally less than half this alcoholic potency, or beer and cider which are less than a quarter. The more concentrated the alcoholic liquor ingested, the more intense the inflammation of tissue. At the same time, an equal quantity of any of the potable alcohols will sooner exhibit its characteristic symptoms if largely diluted with water (Dujardin-Beaumetz and Audigé). Spirits are more maddening than wines and beers, while cider and to some extent beers produce a heavy, stupid intoxication. Absinthe, from the action of alcohol with wormwood, provokes to epileptic seizures, mania, and violence (Magnan). The variety of the alcohol affects the symptoms. Methylic alcohol (wood-spirit) is the most rapid and transient in action. Ethylic alcohol (that of the purest fermented wines) is the least noxious. While butylic, amylic, and propylic, the heavier alcohols, add muscular tremors with severe aches and pains especially in the head, and are more lethal in operation; all the alcohols (Dujardin-Beaumetz and Audigé) are poisons. All alcoholic influence is the same in kind, varying only in degree. The common belief that there is no wine or beer, but only spirituous, inebriety is an error. Of the inebriates treated at the Dalrymple Home in England, 8 per cent. have been wine or beer drinkers. Especially with beer there are apt to be dyspepsia, gout, depression, torpidity of liver, and anasarca, with melancholic suicidal tendencies. In a large number of drinkers more potent sophistications add to the virulence of the original alcohol. Another modification of alcoholic action is by idiosyncrasy. One gets drunk in his head, another in his legs; one becomes boastful, another retiring; one is merry, a second is violent, a third is drowsy.

Alcoholism may be *acute* or *chronic.*

Acute Alcoholism.

Intoxication is the most prominent acute pathological process. That arising from ethylic alcohol is the simplest and least harmful type. The first stage of drunkenness is vascular relaxation, characterized by a glow of warmth and pleasure with exaltation of ideation. The pathology of this exhilaration stage is vasomotor paralysis causing reduction of the nervous control of the vessels, the dilatation extending through the vascular system of the brain. The second stage is one of further diminution of inhibitory control, with cerebral automatism and incomplete partial paralysis of the brain and higher nerve centres. There are disturbance and exaggeration of the emotions. The common belief of *in vino veritas* is, to the writer's thinking, erroneous. The drunken man has an *alter ego*, often the opposite of the sober and normal *ego*, the quiet man becoming boisterous, and the boastful bully a shrinking mute. Incomplete temporary paralysis is seen in tottering gait and thickening speech, with muscular inco-ordination of the upper and lower limbs. In the third stage of dead drunkenness the intellectual faculties are in abeyance, the only spark of vitality smouldering in the heart and circulation.

In some cases, even in adolescence, fatal *acute alcoholic coma* has followed the rapid consumption of a large quantity of spirits, but this issue has resulted from a comparatively smaller quantity in persons exhausted by insufficient food, by prolonged exposure to heat or cold, by want of sleep, by too lengthened excessive toil, and by disease. In a case seen by the writer on December 19th, 1894, a married woman, aged forty-one, died without recovering consciousness in five and one-quarter hours after having at a draught drunk two and three-quarter pints of whiskey. The face was pale, eyes suffused and dull with widely dilated pupils, temperature 91° F., the pulse thin, compressible, and barely perceptible, the breathing stertorous. In slight cases, the comatose person is semi or wholly unconscious, or sometimes dull and drowsy for hours; in grave cases, there are complete and prolonged insensibility, stertorous breathing, pallid face with symmetrical features, no power of movement, slow, weak, compressible and labored pulse; the surface of the skin is cold, moist and clammy with perspiration, the temperature is sub-normal to the extent of from 1 to 6 degrees, and sometimes there is emesis. The pupils may be contracted, dilated, or unequal, and at times there is involuntary discharge of fæces and urine as a consequence of the rectal and vesical paralysis. These symptoms may be complicated with unilateral facial paralysis, epileptic or other convulsive seizures, or apoplexy. The diagnosis is to be made from non-alcoholic coma which is often mistaken for the alcoholic form owing to a spirituous odor arising, even in the case of teetotalers found insensible in the street, from brandy administered by a bystander. Apart from a history of drinking, only examination of the contents of the stomach can determine the presence of alcohol as a causative factor. It has been claimed that when pressure is made upon the supra-orbital notches, compressing the nerve, the alcoholized person immediately shows evident signs of life, and that the effects of alcohol will thus be kept in abeyance for a time. Diabetic and albuminurial coma may be discriminated by an analysis of the urine, but it should not be forgotten that albumin is sometimes present when the coma is due to alcoholism alone. The diagnosis between acute alcoholic coma and non-alcoholic coma from head injury is occasionally called for in the person of individuals found insensible on a public thoroughfare. Waters proposes the removal of the alcoholic symptoms by inhalation of the vapor of ammonia, but the writer cannot recommend this procedure, the risk being too great in grave cases. An apoplectic attack in nowise due to alcohol not unseldom simulates drunkenness. The writer has known abstemious persons, while laboring under a sudden attack of apoplexy, stagger and talk thickly so like a drunken man that they have been treated as if they were simply drunk; and so many cases are on record of the death in a police cell from apoplexy of persons who were arrested for drunkenness, that the heading of "Drunk or Dying" has been a familiar feature in the daily newspapers. A normal or elevated temperature and unequal pupils point to the presence of disease not necessarily of alcoholic origin, but there is no satisfactory diagnostic sign other than an accurate history of the case. So difficult is the diagnosis, that skilled observers, such as hospital physicians, have again and again been mistaken.

The only safe rule in such obscure cases is to treat the patient as suffering from an underlying and grave ailment till the acute alcoholic symptoms have had time to disappear. The London *Morning*

Post of October 20th, 1894, reports the proceedings at a coroner's inquest, held on a gentleman, aged forty-four, who was found apparently drunk on the footpath, who was taken up as drunk by a constable, examined and declared to be drunk by an experienced police surgeon, but who died next day of fracture of the skull. The external application of heat to the lower extremities, when the head feels hot, a cooling lotion on the latter region, and the hourly administration of a little hot water containing some compound cinnamon powder, combine to form the safest immediate treatment in obscure cases. There must be an exception to this rule when there is a suspicion of poisoning by opium (indicated by pin-point pupils), by belladonna (indicated by dilated pupils), or by any other poison, or where there is a suspicion of alcohol in the stomach, when that organ should be emptied by an emetic, stomach pump, or siphon tube. In alcoholic coma especially, it is essential to keep the surrounding temperature a little above blood-heat. Before police cells were kept warm, drunken prisoners sometimes met with their death owing to the alcoholic reduction of the inmate's temperature. To guard as much as possible against error, the London police are instructed to be very careful in dealing with persons found insensible, to call in the divisional surgeon or a neighboring qualified medical practitioner in all doubtful cases, and to visit the detained every half-hour.

A similar error to this mistaken diagnosis of the condition of persons found insensible out-of-doors occurs occasionally in private life. The writer has repeatedly, after a post-mortem examination, had to testify at a coroner's inquest that the deceased had died from cerebral or meningeal hemorrhage, though he or she had been regarded by friends as intoxicated only. R. T., aged thirty-eight, female, died from cerebral apoplexy. The husband and deceased's sister had, twelve hours before her death, found her insensible and "breathing hard," but as they concluded that she was "drunk again," having frequently seen her in apparently the same state before, they simply let her lie as she was over-night, finding her dead late next morning. In many such obscure cases, whether in the open or at home, certain symptoms in cerebral hemorrhage and other non-alcoholic conditions are specially apt to mislead even practised physicians. The odor of spirits, with mental confusion, particularly when the comatose one can be roused for a few seconds to partial consciousness, when accompanied by anger, violence, obscene language, or swearing, has often elicited a hasty but inaccurate diagnosis of drunkenness. Discontent, violence, swearing, and obscenity have been noted in cases of non-alcoholic apoplexy, and in cases of non-alcoholic head injury. Even the presence of a poisonous dose of alcohol in the urine (said by Anstie to be demonstrated when the addition of one drop of urine immediately turns 15 minims of a chromic acid solution of the strength of one part in 300 parts by weight of strong sulphuric acid a bright emerald green) is not sufficient to warrant an opinion of alcohol poisoning only. The coma of alcoholic intoxication is in itself grave, but it may coexist with fracture of the skull or intracranial hemorrhage. As Hughlings Jackson puts it, a correct diagnosis can be arrived at only by exclusion. Especially must disease and injury of the head be excluded. A drunken man may be excitedly intoxicated or he may be so "dead drunk" as to be incapable of excitement. Even the absence of any sign of paralysis cannot be regarded as proof that there is no cerebral lesion. In one case in Middlesex Hospital, the patient, having prior to transference from a police cell been adjudged "drunk," though there were no paralytic symptoms, an autopsy (the patient having died on the thirteenth day after admission) disclosed laceration of brain substance, extensive hemorrhage, and fracture or fissure extending into the lambdoidal suture. Morris (1894) specifies the following abnormal states from disease or injury that may be mistaken for drunkenness: fracture of the skull, concussion of the brain, cerebral hemorrhage, embolism and thrombosis, uræmia, epilepsy, narcotic poisoning, heat apoplexy. The rule ought to be to search for bruises, injuries, depressions in the skull, etc. Among other than alcoholic states of excitement and partial or total consciousness, bromidism should not be excluded.

Acute alcoholic gastric catarrh gives rise to intense gastric pain, emesis, thirst, and distress. The agony is occasionally so acute as to render a diagnosis from acute poisoning by arsenic imperative. In such cases the narcotic influence of the alcoholic poison seems to be overpowered by its irritant action, alcohol being really an irritant narcotic. The writer has had several recoveries where arsenic was at first suspected, and a strong suspicion of arsenic in one fatal case where the man was found dead after drinking heavily. At the post-mortem the inner surface of the stomach was intensely congested, fiery red, with patches of a deeper hue, and altogether presented a very angry and inflammatory appearance extending over the pyloric surface to the duodenum, with thickening of the mucous membrane and exudation of grumous, slightly muco-purulent matter from bleeding points. The injection of the vessels presented an arborescent appearance, though the entire inner surface of the stomach was red and inflamed. These appearances were characteristic of arsenic poisoning, and only a chemical analysis, by showing the absence of arsenic, proved the purely alcoholic source. The alcoholic post-mortem appearances were congestion of the liver, injection of the meninges, cerebral congestion with ventricular serous effusion, engorgement of the lower lobes of the lungs, and distention of the right heart cavities with semi-fluid blood. In the treatment of such cases, the poison is to be at once withheld, hot opiate poultices or fomentations to be applied to the abdomen and dry heat to the lower extremities, if necessary; the stomach to be emptied and washed out with tepid water; milk with soda or lime-water and barley- or rice-water to be administered. If collapsed, cinnamon may be added to the milk, with opium or morphine in pill, or in an effervescing mixture to allay severe pain. Sometimes bismuth is useful. A frequent sequel in non-fatal cases is jaundice, a rarer one pneumonia.

Other forms of acute alcoholic poisoning may specially involve the kidneys, as shown in *nephritis* and *albuminuria* with acute dropsy and convulsions, or the liver with symptoms of acute *jaundice,* or the lungs with *acute alcoholic pneumonia;* though death by nervous shock is the usual fatal termination after a debauch without exposure. Beaumont's observation of the behavior of alcohol on the gastric mucous membrane of Alexis St. Martin disclosed, after a slight excess in alcohol, considerable erythema with aphthous patches and thick ropy mucus tinged with blood. In rapidly fatal alcoholic cases extravasation of blood (pulmonary apoplexy) has been seen (Tardieu) with engorgement of the venous system of the brain, cerebral and meningeal hemorrhages. In this class of cases it is important to clear out the stomach contents, to apply external warmth, to keep the temperature of the apartment well up, and to give bland liquid nutriment (warm milk by preference) and unintoxicating restorative carminatives. Amyl nitrite has been suggested, but the writer has found it uncertain and not nearly so useful and safe as aromatic powder (cardamom, ginger, and cinnamon) in hot water.

Acute Alcoholic Insanity.

Mental disorders of various kinds and degrees are met with in acute alcoholism, from the poisonous influence of alcohol on brain and nerve substance, for which this poison has a strong affinity (Richardson), and may be grouped under the heading of *acute alcoholic insanity.* *Mania-a-potu* is a marked immediate effect of acute alcoholic poisoning. This disease rarely attacks constant inebriates, and an attack is generally ushered in without warning or visible prodromes. The seizures, which resemble those of convulsive epileptic mania, usually occur a very short time after the ingestion of the exciting alcoholic dose in occasional or periodical inebriates, who have spells of temperate repose between their inebriate outbursts. In the few instances where this mental riot affects continuous inebriates, the neurotic storm is most easily provoked after an interval of abstinence, though the writer has known cases in which the drinking of more than a certain quantity of liquor invariably brought on an attack. This is a mania of violence, a maniacal delirium, with little or no muscular tremor or hallucination. There may be complete unconsciousness of surroundings, the infuriated subject being literally "possessed," for the time being utterly beyond his control, and though sometimes hurled into homicidal violence, generally with no remembrance of his violent acts. The paroxysms of violence may be restricted within a few minutes, or may last for hours or days with quiescent intermissions. An extremely rapid, strong, and full pulse is succeeded, in the calm after the storm, by a steady, quiet, and normal pulse, with no alteration of temperature. Sometimes this variety of alcoholic insanity assumes the form of pure pugnacity, the temporary madman "spoiling for a fight," and exhausting himself in pounding his wife, smashing furniture, throwing everything out of the windows, or trying to batter down a stone wall, break a window, or deal blow after blow in ungovernable rage at an animal, a vehicle, or a ship. Sometimes the affected simply raves incoherently at the pitch of his voice. At other times he is a prey to homicidal impulses. A goodly proportion of police court cases of drunken violence are subjects of mania-a-potu.

Complications are rarely present and recovery is in general rapid. There is little difficulty in diagnosis from delirium tremens, the tremor, terror, and hallucinations of the deliriate being a perfect contrast to the muscular contraction, audacity, and blind rage of the maniac. The white tongue, nausea, sickness, and busy delirium of delirium tremens are absent. Mania-a-potu may follow the ingestion of a small quantity of alcohol, delirium tremens hardly ever occurs except from large quantities taken in rapid succession or smaller quantities long continued. It has been observed that mania-a-potu has appeared in persons who have had a congenital tendency to mental excitement and instability, or in such as have suffered from acquired mental impairment as a result of alcoholic or other intemperance, of traumatism, or of brain-fag. The nervous system of individuals liable to attacks of mania-a-potu, whatever the origin of the diathesis, appears to be responsive to an amount of excitation which would have no appreciable influence on the bulk of mankind, and it is very probable that some of such persons, if free from alcoholic excitation, would be subject under provocative conditions to those epileptic maniacal explosions which so closely resemble the explosive phenomena of mania-a-potu. A simple non-alcoholic liquid diet, with saline draughts in combination with small doses of the bromides and ipecacuanha, and at times cold affusion, constitute the most satisfactory treatment. In prolonged paroxysms a wet pack is useful where it can be applied.

At times, *acute alcoholic melancholia* with or without a strong suicidal tendency is suddenly developed. Under judicious treatment by nerve and liver tonics, such as strychnine and nux vomica with euonymin, by air exercise and cheerful environment, intoxicants being strictly forbidden, there is speedy recovery. If necessary, forcible feeding must be resorted to. The prognosis in general is favorable.

Delirium tremens, though in the greater number of cases occurring in inebriety of considerable standing, occasionally attacks a limited drinker after or during a single drinking-bout. In certain neurotic individuals this disease sometimes arises after a first night's debauch, a direct form of acute alcohol poisoning. The writer has seen it as a result of beer and wine indulgence, but it is mostly the outcome of spirituous excess. Though imperfectly rectified spirits with their fusel oil and other heavy alcoholic crudities, and sophisticated wines, are more apt to incite to delirium tremens, he has seen it arise from the consumption of the purest intoxicants. The exact physical conditions giving rise to this disorder have not been elucidated, but some persons are specially liable to be attacked. The writer has seen limited and unlimited drinkers living under fairly similar conditions as to alcohol-taking and other circumstances, yet while the majority, though suffering from some other ailment of alcoholic origin, never had a single attack of delirium tremens, a small minority of somewhere between two and three per cent. were repeatedly attacked, the number of attacks ranging from one to seven. It was, till Laycock exposed the fallacy more than a quarter of a century ago, formerly believed that cutting off the liquor suddenly brought on an attack, and that "a hair of the dog that bit him" was essential to successful treatment. We now know, however, that abstinence is not the cause, and that the disease is the issue of a material poisonous influence on the nervous system, probably on brain and nerve substance. Though to the taking of alcohol alone an attack can often be traced, many forms of nerve exhaustion, as from physical or mental overwork, starvation, mental perturbation, the shock of an injury or an operation, or the onset of some acute illness, appear to have an influence in precipitating an attack of delirium tremens.

Sometimes the patient is seized without any premonitory symptoms except a disinclination for food, though in a few cases even this has not been apparent, the sufferer having gone to bed seemingly quite well and having awoke during the night cowering, trembling, and terror-stricken, a prey to reptilian or other more loathsome illusions of living things about person or bedclothes, with characteristic tremors, tongue, countenance, and pulse. In general there is a preliminary stage extending from a few hours to several days, of restlessness, distressful uneasiness, and anxiety, disturbed sleep, if any, suspicious fear, and distaste for food. As the attack advances, it is accompanied by a tremulous whitish-coated tongue, and a cool skin, moist and clammy with perspiration, most marked in the upper and lower extremities. The pulse is soft, compressible, feeble, and frequent. With the aggravation of these symptoms, often from the first there are terrifying ocular illusions of mice, rats, snakes, reptiles, birds, wild or domestic quadrupeds, from which the patient constantly tries to escape, or which, in his despair, he vainly tries to clutch. There is a risk of suicidal attempts during the attack from the intensity of the terror, and fatal accidents now and then happen, as by jumping out of a high window, in the struggle to escape. There may also be auditory hallucinations, the deliriate hearing voices, imaginary people talking about him in the room, constables and others pursuing him for some breach of the law, though these last-named symptoms are oftener seen in acute alcoholic insanity. He keeps muttering and talking incoherently and incessantly night and day, often about his ordinary vocation. There is no intolerance of light simply because the trouble is primarily nervous and mental. If the attack does not subside in from two to four days the pulse becomes more frequent, thin and thready, there are a brown-coated and dry barked tongue, and subsultus tendinum, with continual muttering delirium and picking of the bedclothes or arms, the patient dying either from exhaustion or in a violent paroxysm.

There are three distinct forms of delirium tremens. The traumatic variety may arise altogether independently of any alcoholic local brain lesion, from neurotic shock consequent on some injury by accident or violence (more common after accident than violence), or on some disease (alcoholic or non-alcoholic). As a rule traumatism excites the disease in steady more or less moderate or immoderate drinkers, regular twice or thrice a day drinkers whether limited or excessive, who have their nervous system, blood, and circulation so continuously under the influence of the alcoholic brain and nerve poison that, though they can carry on vital functions under ordinary conditions, the nerve centres are unequal to a sudden demand for an unusual supply of nerve energy. Yet once and again, symptoms undistinguishable from ordinary delirium tremens, except for the exclusion of a history of alcohol, have occurred in most abstemious persons. These cases, however, are very rare, and there is almost always a family history, as in the case of a merchant who at thirty-one, probably from excess in tobacco, though a life abstainer, shook with general tremors every morning till a pipe steadied him. His father had been a chronic inebriate. This may also be said of a form of imitative delirium tremens occasionally appearing in young people, generally characterized by muscular inco-ordination and fatuity of gait and speech (by the last two symptoms at once distinguishable from chorea). Though both of these classes of non-alcoholic imitative delirium tremens are rare, their possibility should not be forgotten, as an important medico-legal question might depend on the true origin of the symptoms. A familiar illustration of the traumatic form is often afforded in general hospitals in large cities, where brewers' draymen and other steady hard beer-consumers develop a violent attack of delirium tremens soon after their admission for very slight injuries.

Prognosis.—Idiopathic delirium tremens may be of a mild or of a grave type. In the less serious form, though there is the characteristic busy delirium of wakefulness and fear with hallucinations and delusions, with bloodshot eyeballs and tremors, the temperature does not rise to 100° F., the symptoms fade in from three to five days, and in a few days more the patient is apparently quite himself again in bodily activity. In the grave form the prognosis is serious even in a first attack, the characteristic symptoms are more pronounced, the temperature keeps several degrees above normal, the sufferer is more unmanageable instead of showing a tendency to recovery, the pulse becomes quicker, weaker, and thinner, and general prostration sets in, deepening *pari passu* with the violence of the acute paroxysmal, convulsive, and uncontrollable attacks; the tongue becomes furred, the fauces and throat parched, till convulsions or exhaustion terminate the struggle. If the attack is inaugurated by convulsions or these occur in the progress of the attack (as the writer has frequently seen), gloomy prognosis is heightened. The prognosis of the milder idiopathic form, and also of the traumatic, is favorable. In all the forms, the occurrence of previous attacks and the presence of any concurrent disease, as fatty degeneration of the heart, kidney disease, or cirrhosis of the liver, adds to the gravity of the case. Yet, all round, the writer has seen recovery in over ninety per cent. of cases. Delirium tremens is oftener met with among males than among females, the proportion of the latter in the writer's experience having been about fifteen per cent. of the whole number. Delirium tremens appears to have the predominance among men that alcoholic multiple neuritis takes among women.

The *diagnosis* from mania-a-potu has already been dealt with.

That from the delirium of chronic alcoholism can be made from the lighter tremors, and the absence of the white tremulous tongue, thirst, feverish symptoms, great bodily disturbance and acute fright of the latter disease. Delirium tremens may be discriminated from the delirium of pneumonia and of some forms of non-alcoholic cerebritis and meningitis, and from the muttering delirium of typhus and typhoid, by the history and progress of the attack, and by the absence or presence of other symptoms like the rash of typhus, the diarrhœa, abdominal pains, and spots of typhoid, and the dulness of the lungs on percussion with the other physical signs of pneumonia. In phrenitis, the quantity of phosphates in the urine is diminished, but in delirium tremens increased. Care should be taken to discriminate a form of absinthal delirium tremens described by Collet as occurring in the person of a man, aged fifty-nine, residing in Lyons, who appeared at the hospital. There was neither cerebral excitement, nor hallucinations, nor nystagmus, nor trembling of the tongue, but there was general tremor with reflex hyperexcitability.

Treatment.—Of the treatment there has been no end. Not so very long ago, all cases were freely plied with alcoholic beverages from outset to finish. The question used to be, not "Shall this patient have an intoxicant?" but "What stimulant shall he get?" the practice being to give liberal quantities often of both wines and spirits. During the past thirty-five years or so, the superstition on which this treatment was based, that it was perilous to cut off alcohol at once, has gradually been exploded, though the writer has now and then met with patients whose attending practitioner has shrunk with horror at the risk involved in "so dangerous a step." By respectable authorities we are even now gravely assured that in serious cases it is imperative to continue to give the patient the same intoxicating drinks in which he has been accustomed to indulge, and that it is a mistake to suddenly withdraw the liquor. An experience of some hundreds of cases has shown the writer that this is an erroneous belief. In this disease he has never seen the liquor do anything except decided harm, in some instances putting in jeopardy the safety of the patient, in other instances contributing to a fatal issue. Both by their direct effects and by their influence in reaction, alcoholic drinks are injurious. The patient is laboring under a toxæmia induced by alcohol, and to keep giving him more of the poisoning agent is to keep up the toxic action. He is being weakened by the excessive and prolonged discharge of nerve energy, and by his muscular convulsive restlessness and struggling. If more alcohol is supplied, more nervous energy is called forth and there is more muscular excitement. Thus he is directly enfeebled. Further, after alcohol freely administered there is apt to be reaction, which reaction in this and other diseases not unseldom perils the recovery of a worn-out and collapsed patient. There need be no fear of serious danger from the sudden cessation of stimulants. Not many less than one hundred thousand criminals and paupers are admitted into prisons or workhouses every year in Britain. The bulk of these are broken-down inebriates shattered by alcohol, yet their liquor indulgence is forthwith cut off, not only without injury but with such remarkable benefit that they speedily regain health and strength. The first condition of cure, then, is complete abstention from all intoxicating drinks. Where any risk is feared, aromatic spirit of ammonia in cinnamon water, aromatic powder in hot water, or hot water itself, or some liquid preparation of beef, or hot milk or broth may be substituted with advantage. It is true that even in some special institutions for the treatment of inebriates, beer, stout, or an ardent spirit is employed in small doses as a calmative; but, though this quieting is sometimes achieved for the moment by the anæsthetic influence of alcohol on some individuals, it is never unaccompanied (even when for the moment successful) with some risk of subsequent damage. The writer therefore strongly insists on the exclusion of intoxicating liquors from the remedies with which we should attempt to combat delirium tremens. In incipient slight attacks, soon after the premonitory symptoms have threatened a seizure, a good dose of tincture of opium, an old-fashioned but useful drug, will sometimes produce a sound long sleep and avert the threatened attack. With this exception, the less opiates are resorted to, the better. The once common free administration of opium with alcohol has been proved to be as unsafe and unreliable in delirium tremens as it is in cholera. Opium and morphine in substantial doses, by their lowering of vital energy diminish the chances of recovery, apart entirely from the risk of an overdose. It seems to be too often left out of consideration that opium and morphine do not counteract the deleterious narcotic action of alcohol, but rather intensify it. That the risk of an overdose of opium is real, the reports of many inquests testify. Fellow-students of the writer, fairly moderate regular consumers of alcoholic intoxicants, have taken to opium or morphine to secure the sleep which their arduous labors as physicians or scientific investigators have robbed them of, and have been found dead from the combined effects of the two narcotics; while patients laboring under an attack of delirium tremens have died from a subcutaneous injection of an opiate administered by the attendant physician. Digitalis, in one dose of half an ounce of the tincture, followed if necessary by a second in a few hours and a third of two drachms, has been highly recommended by Jones; but the writer found it of no avail in averting a fatal issue in one case, and its potency demands great caution in its administration. Opium used to be largely employed in the endeavor to induce sleep, which has been deemed by many to be the true cure of this disease. But there are two kinds of sleep: one, sound, healthful, and natural, which, if sufficiently prolonged and if the patient awakes refreshed and calm therefrom, is the principal curative process; the other light, disturbed, fitful, short, and morbid, on coming out of which the patient awakes unrefreshed and delirious or semi-delirious. The sleep secured by opium and morphine seems usually to be of the latter variety, and in many cases, by the subsequent reactionary disturbance, to aggravate the nervous tumult. These drugs require to be given in very large doses to procure sleep in delirium tremens, and with such heroic doses, in tissues already saturated with alcohol, deep and prolonged coma may supervene with a fatal issue accompanied often by profuse serous cerebral effusion. If an ordinary dose of either narcotic is given, the brain is only excited and the general nervous perturbation intensified. In one case of considerable gravity in a male intemperate of the age of twenty-seven, the writer tried the experiment of an evening dose of Dover's powder one day, and on the succeeding day an emetic dose of ipecac. The sleep following the Dover's powder was restless and disturbed, the patient next morning when fully awake feeling parched and excited; while after the emetic of the preceding evening he had a sound, restful, uninterrupted sleep of at least five hours' duration, and awoke comforted, rested, and refreshed, with a steadier hand and a quieter brain. A good deal of these unfavorable effects may also arise from cannabis, chloral, sulfonal, ether, chloroform, hyoscyamine, and to a less extent from the bromides. Phenacetin in 8 to 10 grain doses for not more than two hourly repetitions is less risky, but is unreliable. Chloral and sulfonal are depressing to the heart, besides their lowering and disturbing influence on brain and nerve function, the latter having in many cases a strong and somewhat prolonged injurious depresso-excitant effect on the cerebellum, the patients sometimes for days or weeks having a tendency to fall backward, and to walk with unsteady gait. Paraldehyde, trional, and other hypnotics, less dangerous than opium or morphine, are all more or less liable to be succeeded by reactionary excitement after the artificially induced sleep is over. Potassium and sodium bromides, either alone or in combination with chloral and henbane, have certainly at times proved useful; though even when given by themselves, they have often, to be effectual, to be given in such very large doses, or in so many repetitions of a dose (by the summation of their cumulative influence more than equivalent to such an unusually large dose) as seriously to depress vital function and deprave the blood. Even such comparatively innoxious antispasmodic drugs as asafœtida and such harmless tonics as calumba and humulus, though productive of no direct injurious effects through their own action, and of no substantial reactionary after-symptoms, are open to the objection that reliance on them is wasting valuable time. The writer excludes such more potent tonics as strychnine and its preparations as out of the question till the delirium has ceased.

As delirium tremens is a direct product of alcoholic poisoning, practically an acute disease produced by the action of the poison on a nervous system so responsive to such acute poisoning, either by reason of a preceding chronic poisoning or of a susceptible nervous organization rendered so by a constitutional neurosis either inherited (as it generally has been) or acquired under abnormal non-alcoholic conditions; as, in fact, the delirium is simply a symptom of active poisoning, the first indication of treatment is to eliminate the poison as rapidly as possible. None of the medicinal substances just enumerated can take an active part in this process of elimination, however efficacious they may possibly be in other respects. The poisoning process must be grappled with by speedily freeing the system from the poison, and there are but three easily available means of achieving this end. So far as this alcohol elimination is concerned, the object might be gained by diaphoresis in a Turkish hot-air bath, but the depression and exhaustion consequent on a sojourn long enough to be effectual, as well as the impossibility of securing a bath with adequate attendance, in most cases, contribute to render this plan as a rule impracticable. Even in a hospital furnished with an ample Turkish bath, expensive attendance would be needed, the temporary dwellers in the sudatorium being bodily unrestrained by bedclothing, with the additional risk of the deliriates injuring themselves in their convulsions and struggles. The perspiration which is the only efficient method of securing the necessary elimination of the poison might be secured by the wrapping of the patient in a wet pack, but this is liable to the special objection that, particularly in grave cases, the close restraint of the contorted sufferer in a properly fitting pack would heighten the nervous excitement and cerebral pressure, every one knowing this to be the effect of forcibly restraining violent involuntary muscular action. In light forms of delirium tremens, both the Turkish bath and the wet pack are sometimes of service where they can be conveniently applied, and the writer will later on speak of their great usefulness in certain phases of chronic and subacute alcoholism; but in violent cases of delirium tremens he cannot recommend either, in ordinary practice.

In addition to rapidly clearing the alcohol out of the body, it is of the highest importance to conserve the patient's strength. To restrain his muscular contortions would be more quickly thereby to exhaust his vital energy, which, especially in those who have had repeated attacks and have not been abstemious in the intervals, is not generally so abundant as to warrant any preventable loss of it. This preservation of nerve force is best accomplished by allowing the muscular movements to be as untrammelled as possible. There are two ways of providing for this. One is by shutting up the delirious patient in a padded room, where he can have nothing to injure himself with and cannot hurt himself, even if he dash himself in blind fury against the padded walls. This is a procedure which is rarely obtainable, though by the favor of the master of a London workhouse the writer was once enabled to employ it in the case of a publican, who could not be properly controlled or fed in his public house by five men, and whose unruliness and cries were seriously detrimental to the trade carried on in the shop beneath his bedroom. The man received no medicine, milk and coffee being handed to him through an aperture in the door padded in the interior, but in some three days he exhausted himself, and fell into a profound and long sleep, from which time he steadily and thoroughly recovered. This excellent provision of a padded room being, however, rarely within our reach, must be regarded as in general practice also impracticable. One is therefore driven to find a medicine which, while operating, as soon as may be, to get rid of the accumulation of alcohol in the system, would not by the depression of narcotization or reactionary excitement weaken the *vis medicatrix*, in which latter really lies the potency of cure. All this the writer believes he has found in the innocent yet powerful liquor ammonii acetatis, which in addition seems (either by its antifebrile properties or by some unknown chemical action on the alcohol-saturated tissues) to exert a specially favorable influence on the rapidity and completeness of the elimination. The usefulness of this drug in alcoholism is not limited to delirium tremens; but in delirium tremens it has appeared to combine all the advantages of the cooling and calming emetic treatment by ipecacuanha, with, by the characteristic profuse perspiration which it causes, the best qualities of the wet pack and hot-air bath. Withal, there being neither chemical nor muscular restraint, the restless sufferer's already lessened nerve force (lessened by the disease) is not further drawn upon to combat the depression occasioned by opiate or other narcotism, and the nervous exhaustion from the excitation aroused by the physical restraint (envelopment in a close packing of sheet and bedclothes, as in a strait-jacket) of turbulent and inco-ordinate muscular contractions. Since relying mainly upon the acetate of ammonia, the writer has had much less trouble with cases of delirium tremens, a great deal less anxiety from serious complications (which formerly chiefly arose from the after-effects of opiate, chloral, and bromide remedies), and a much larger percentage of good recoveries. Of course, care must be taken to have sufficient attendance to guard the patient from harm. This is a point very often of vital importance. If in delirium the vigilance is relaxed, there may be a sudden termination of the case. Though, usually, alcoholic deliriates in attacks of delirium tremens are fairly easily manageable, suicide may be attempted in almost the momentary absence of a nurse. The maximum extent of freedom compatible with safety is attained by the patient being kept in bed. With a little tact, an intelligent attendant can exercise considerable control over the excited patient by pressing down on the patient near and over the knees outside the blankets. This allows free play for practically all needed involuntary muscular movement. The patient is thus allowed to exhaust himself by his continuous muscular efforts, till the true restorative state of natural sleep overtakes him through sheer weariness. There is nothing else so valuable here as the sound, wholesome sleep of fatigue; in character and effects so opposite the unwholesome, disturbed and disturbing sleep procured artificially by narcotics. In all that has just been stated about the treatment of delirium tremens, and this is equally true in other diseases, it should ever be borne in mind that exceptional cases may occur which may require alcohol or other remedies not here referred to. The writer is ready, for example, to administer alcohol at any time when it appears to be called for, but he has never yet seen a case of delirium tremens in which he considered this remedy was desirable.

Acute Alcoholic Epilepsy.

A form of acute alcoholic poisoning, closely allied, by its nervous character, to the forms of acute insanity induced by alcohol, yet never amounting to actual insanity except in its form of epileptic mania, is acute alcoholic epilepsy. Though, like delirium tremens, it may arise from time to time in the course of chronic alcoholism, it frequently is an early manifestation of acute alcoholic toxication. Alcoholic epilepsy presents itself in two forms, one unaccompanied, the other accompanied, with maniacal phenomena. The simple form without mania is characterized by the very common and well-known epileptic explosions, which are familiarly designated epileptic convulsions or fits, during which the afflicted is unconscious, feeling no pain though the subject of rhythmic violent muscular contractions. The mind is a blank, but as there is no mania the patient, though contorted with convulsions, is passive. In the complicated form, when the convulsions have ceased, or in the intervals between them, or immediately before their appearance, the subject of epileptic mania may perpetrate criminal deeds of violence though with no consciousness or memory of their perpetration. As we have already seen, the latter form is frequently developed by indulgence in absinthe (alcohol with wormwood); but it is also provoked by ordinary, especially the stronger, spirituous liquors. There is a question as to whether there is such a special ailment as alcoholic epilepsy. The writer has come to the conclusion that there is, at least of the maniacally complicated form. When of alcoholic origin or excitation, the symptoms have seemed in many cases to have been very similar to those of mania-a-potu (which is unquestionably of alcoholic birth), with a greater tendency to that homicidal mania which Garnier has observed in Paris in the adolescent of alcoholic parentage during the past decade, indicative of that marked psychological as well as cerebral degeneration, which seems to be mainly due to alcoholic physical degradation, arising especially in France from the increased consumption of the heavier alcohols. Whether this belief in a special alcoholic form of epileptic mania will ultimately turn out to be tenable, the fact that the explosions of many epileptics are practically directly attributable to their personal alcoholic excess warrants our special consideration of this condition here. In different instances the writer has observed that non-maniacal and maniacal epileptic paroxysms are in evidence only when the patient has been freely indulging in spirits, or in the stronger wines. When the liquor has been given up, for weeks, months, or years there have been no epileptic symptoms, but these have reappeared very soon after a resumption of drinking. It should be noted that the drinking has not been always to drunkenness. In some of the cases, what would be called a "very moderate" allowance has sufficed to cause the convulsions alone in one person, and mania with convulsions in another. In some cases a permanent cure has been brought about by persistent strict abstinence from all intoxicants, but from his own professional observation the writer should expect, even in these recoveries of from six to twenty years, an early reappearance of the former convulsive and maniacal symptoms, if even the "moderate" use of intoxicants were resumed. In some of these cases there was a history of either an epileptic or other neurosis, in other cases there was no family history of nervous disease of any kind, and the paroxysms seemed to be the immediate effect of a poisonous action on the nervous system.

Though he has seen epileptic attacks traceable to lead-poisoning alone, when there was no serious pedigree or personal history of alcoholism, the writer has never met the maniacal complications, only the simple epileptic muscular contractions, and he has never seen these spasmodic epileptic movements result from acute, they having always appeared in chronic, lead poisoning. This affords one diagnostic mark between plumbic and maniacal alcoholic epilepsy. Further, epilepsy apparently due to alcohol is more often met with than epilepsy apparently due to lead. The history of the patient prior to the attack and the progress of the epileptic symptoms will help still more to discriminate between alcoholism and plumbism. The tendency to recovery is very much greater in alcoholic than in plumbic epilepsy, and the characteristic appearance of the gums is generally more or less marked. Death by coma after a number of seizures, occasionally after only a few, is no infrequent termination; the average career of an alcoholic epileptic extending to from eleven to fifteen years. The potassium, sodium, and ammonium bromides in small doses, in combination with tincture of belladonna in 3-minim doses and aromatic spirit of ammonia in doses of 8 minims, in a tonic infusion internally, with gentle purgation by Hunyadi János or some other similar mineral water, have proved the most serviceable remedies in the writer's hands. The nutriment must at first be simple, digestible and fluid, and alcoholic intoxicants must be vigorously excluded. In maniacal cases the patient must be closely watched. In these cases small doses of croton chloral or chloral hydrate usually add to the efficacy of the medicines already indicated. After the attack has passed quite away unconditional abstention should be plainly advised as the essential factor in prophylaxis.

Acute Alcoholic Paralysis.

This is now recognized as a distinct group of symptoms of the alcoholic toxic process on nerve substance and texture. Altogether apart from the more chronic varieties of alcoholic paralysis, alcohol being a potent paralyzant only produces in the human constitution what might naturally be looked for from it, when it gives rise to a more or less rapidly generated acute and characteristic paralysis (Lettsom, 1789; James Jackson, 1822; Chomel, 1828; Magnus Huss, 1849; Duménil, 1864; Lancereaux and Leyden, 1881; Oettinger, Hadden and Dreschfeld, 1885; Buzzard and Gowers, 1886; Pearce and Finlay, 1887). Head symptoms are rarely manifest at the outset, and when present there is only occasional headache with a vague undefinable uneasy feeling. There are usually at first languor and lethargy or irritability and anorexia, with disturbed and less sleep. There is often no painful spot, though energy seems to be failing, everything becoming a burden. Often there are slight feelings of numbness in the arms and legs with a sensation of dragging of the toes, momentary attacks of vertigo with an evanescent staggering. All these symptoms may be so little pronounced as to suggest no resort to medical counsel. In some cases all that thus far in the progress of the disease has been experienced has been gastric disturbance, with cramps and various occasional fleeting pains and slightly troublesome prickings gradually referable more and more to the limbs, especially the lower. Such patients when under treatment have frequently been treated only for dyspepsia and its complications. Even the increasing disturbance of sleep, with the discomfort and irritability of temper which slowly herald the approach of the toxic neuritis, have been in the past very naturally set down to the gastric disorder. The loss of power may occur all at once and unexpectedly, no suspicion of any toxic process having been entertained; it appears most often first in the feet, legs, and thighs, then in the upper extremities, especially in the extensors, but in some instances in the extensor muscles of the wrists first. In other cases the paralysis attacks both the upper and lower limbs simultaneously, or almost so. However, in the majority of cases the paresis comes on more slowly, and there is a further development of preliminary trouble before the appearance of actual paralysis. The onset is prolonged by the increased severity of the restlessness, insomnia, tingling, burning, lancinating pains, and muscular cramps, the acuteness of the aches and pains by their intensity driving many of the sufferers (in spite of strict medical injunction forbidding intoxicants), in private cases, to bribe servants and attendants to smuggle in liquor to dull the senses so as to procure some relief from the unbearable agony. Where power in the extremities is not suddenly lost there is a gradual inability to get about, so gradual that it is often impossible to define the daily loss of motor capacity. In favorable cases when the physician has been called in time and has succeeded in effecting complete withdrawal of alcoholic liquor, the disease may be arrested before the lower and upper extensors are seriously affected; but this is very rarely the case. The mischief has been done in most persons before the doctor has been summoned. There may then be double foot and wrist drop, or double drop of feet or wrists alone, or more seldom the drop of wrist or foot may be unequal, never in the writer's experience unilateral. The muscles involved are wasted, loose, and flabby. In the earlier stages there is in some cases, and in others throughout, very little difference in muscular electric reaction; but in most, as the wasting and paralysis become more manifest, the faradic practically ceases, and the galvanic is so faint as to respond slowly only to a strong current. There is hyperæsthesia of the skin with extreme tenderness from the lightest touch on the part affected, the agony caused by the most impalpable contact with the skin over the track of the painful nerves, or on any movement involving the nerves of the implicated muscles, being so intense as to compel the patient to scream. The shooting and tearing pains so acutely increased on muscular or tendinous movements are almost intolerable, the patient often sobbing, crying out, and being afraid to change the position from the suffering this would insure. The pains are more acute at night. Knee and allied reflexes are in most cases diminished or lost, but the cutaneous are sometimes exaggerated, especially over the abdominal region, unless anæsthesia has set in. The anæsthesia is usually more marked in localized areas over individual affected nerves, and there is often a feeling of coldness to the touch. The extremities and their neighborhood, especially the feet and hands, in advanced cases (though not always) are apt to appear smooth and glistening with sometimes a violet hue, with no subcutaneous effusion, with œdema especially over the malleolar regions. A considerable proportion of cases develop ataxic symptoms. These symptoms when present appear usually in the lighter forms of the disease, and are themselves generally slight. The writer has never seen incoördination so emphasized as to give the patient the characteristic gait of a locomotor ataxic. Bastian states that such cases are now spoken of as *pseudo-tabes*, when in combination with pains in the limbs, loss of the knee-jerks, unsteadiness when eyes are closed, and a peculiar gait; and that though this chain of symptoms may be met with in multiple neuritis from any other cause, they seem to be specially frequent in the alcoholic form of the disease. The writer has frequently noted these symptoms slightly developed apart from any other definable disease, in the person of limited and free alcohol-takers. Very rarely, chiefly in protracted cases, where this has not been guarded against by intelligent measures, the fingers are drawn and contracted, as are still more rarely the extensors of the leg. It is usually only in very protracted and intractable cases that bedsores threaten. The writer has never seen incontinence of urine or of fæces. Brain symptoms have rarely been prominent in the writer's experience. Still they have been present to a slight extent in many, especially female cases. Irritability with a tendency to melancholia, failing memory, diminution of the capacity to concentrate thought or to follow any train of reasoning and lessening interest in external objects, have been the chief features in the earlier stages of the disease. Occasionally there may be a transitory delirium, but the writer has not seen this extend over more than a day or two, and only in a few cases. As the disease advances, the patient sometimes (in other cases from the first) seems to be half asleep, and replying automatically as it were to questions. He feels as if in a dream, almost in another sphere, and when his attention is attracted, which is sometimes only with difficulty, he feels as if being recalled from a long way off. This is practically a state or variety of semi-somnambulism, the senses being dulled, the patient feeling so lethargic that he has no mental or bodily energy. He feels and appears dazed, as if in a trance from which he is momentarily and half withdrawn only after some minutes' attempt to rouse him. When aroused he can eat or answer only in a slow, heavy, dreamy manner, and soon relapses into his deep lethargy, with its sluggish, confused, disordered yet on the whole pleasant imaginings. There are various degrees of this semi-trance state, and the writer has never seen the inclination to activity and violence which one sometimes sees in other varieties of alcoholic trance. Only an attenuated degree of this peculiar condition of idle repose is met with in most cases. The degraded *morale* which has been described by some writers as specially characterizing alcoholic neuritis, has not appeared to the writer to present the symptoms of anything beside the usual effects of continuous and excessive alcoholic indulgence. The utterly false accounts which the patient often gives of his asserted temperance are common to various phases of chronic as well as acute alcohol poisoning, especially of the chronic. Indeed the writer has heard fewer false statements of habit, as to alcohol, from sufferers from acute alcohol paralysis than from alcoholomaniacs generally. He accounts for this by the fact that patients suffering from acute alcoholic multiple neuritis have been usually a shorter time addicted to intemperance than ordinary chronic alcoholics, who have escaped this form of toxic alcohol poisoning. In recovery, these somnambulistic symptoms very gradually fade as the poisoned nerve tissues regain their tone. The optic nerve and the eye are rarely implicated, though in a few cases there are amblyopic and other disturbances of sight. The state of the pupils varies, they being sometimes equal, sometimes unequal. The duration of the disease ranges from four weeks or a little less to between three and four years, the average length, taking all cases favorable and unfavorable, being about eight months.

The *diagnosis* from several other affections (principally of the spinal cord) is not always apparent. For a time till the specific symptoms of alcoholic neuritis are developed, it may be discriminated from general paresis by the absence of paralysis of the tongue and lips, and of the grandiose delusions often ushering in general paralysis. The writer has never seen the alcoholic neuritic giving orders on a lavish scale for the execution of works or the purchase of carriages, horses, furniture, and other things far beyond his means, as he has observed in patients in the preliminary exaltation stage of general paralysis. Yet in some cases, before the foot-drop or wrist-drop, so characteristic of alcoholic and saturnine poisoning, has shown itself, experts are for a time apt to be misled as to the actual origin of the malady. Where dropped feet or wrists are accompanied by wasting of the extensor muscles of the limbs, sensations of tingling, a general numb feeling, acute stabbing or tearing pains in different parts of the body, especially the lower extremities, at times darting up the back, articular hyperæsthesia, all objects feeling cold on touching the skin, extreme tenderness on pressure over the limbs involved, following up the track of the adjacent important nerves, with lessened or lost knee-jerk, with the diminished electrical reactions of degeneration, there is fair evidence of multiple neuritis. If in addition there have been such premonitory symptoms as disordered tactile sensibility of the extremities popularly known as numbness of the fingers and toes, vasomotor spasm of the extremities described by the patients as deadness and coldness of the fingers and toes, with distressing cramps in the calf of the legs, which have attacked the sick one suddenly, while falling to sleep, awaking in the morning, or moving the extremities as in using a sewing-machine or writing (these cramps may have occurred weeks, months, or even years before paralysis has manifested itself); or if there have been slight and delicate tremors of the fingers, with generally harmless hallucinations and illusions commonly referred to as "silly," because the patient talks somewhat incoherently and foolishly about actions which he erroneously thinks he has been performing, or places where he thinks he has just been, or work which he has just been doing,—then there is a strong presumption that we have to do with alcoholic multiple neuritis. The greatest difficulty lies in the fact that patients and their friends are generally reticent as to the presence of alcoholic excess. But gradually the symptoms become too distinct to leave room for doubt. In some constitutionally neurotic persons a continuous small quantity of alcohol can give rise to symptoms of acute alcoholic paralysis.

A diagnosis from *plumbic paralysis* can be made by the absence of the blue gum-line, though in many cases even ankle and wrist drop are the effect of lead poisoning alone, and in other cases the joint product of alcohol with lead. So much alike are many of the symptoms and much of the course and termination of the paralyses resulting from lead and from alcohol, that unquestionably not a few fatal cases of alcohol poisoning have been in the past mistakenly credited to the metal instead of to the spirit. Half a century ago hardly any one realized that there was such a toxicity as alcoholic paralysis. There is one striking diagnostic mark, that in alcoholic paralysis the bulk of the affected recover, in the lead form very few.

The alcoholic ataxia of Dreschfield seems to be practically the alcoholic pseudo-tabes of Bastian, and the ataxic form of alcoholic paralysis of Thorain (1894). It is not a common variety of alcoholic multiple neuritis and is seen usually only as an alcoholic, arsenical, or diphtheritic paralysis. The diagnosis here is from locomotor ataxy. Some have relied on "high stepping" as a diagnostic sign of alcoholic paralysis. Owing to the loss of power in the flexor muscles of the ankle, the toes hang down, causing the patient to raise the heel, the sole being seen from the back; in locomotor ataxia the posture of the toes is exactly the opposite, they being raised. In the alcoholic the patient comes down on his toes, in the non-alcoholic ataxy he comes down on his heel, and as the paralysis of the latter usually is not so symmetrical as that of alcohol, more unevenly and with something of a jerk. When the "ankle drop" is distinct this steppage gait is a good distinguishing mark, but where there is little of such a "drop," when the flexor paralysis is not apparent, it need not be looked for. Again in locomotor ataxia there is not generally the history of alcoholic indulgence unless as a remote cause, as in the case of a solicitor in Scotland, aged forty-two, in whom the locomotor ataxic symptoms slowly and imperceptibly came on, when he had become most abstemious, after some twenty years' regular daily drinking of about eight glasses of whiskey toddy; while in the case of alcoholic neuritis he would have been attacked while intemperate and would have improved almost as soon as he gave up strong waters. Still further, in locomotor ataxy there commonly are the Argyll-Robertson pupil phenomena, the reaction of degeneration, and bladder troubles from time to time with diminution of sexual desire and capacity. Syphilis itself is a factor, sometimes the only factor in the causation of multiple neuritis and of locomotor ataxy, as well as of general paralysis. Though alcohol is frequently associated with syphilis in the appearance of all these three varieties of nervous disease, being a poison which affects the neurin, nerve cell, and nerve function, it is of importance where possible to correct the syphilitic morbid causative condition apart from the alcoholic in such conjoint paralyses. And where either syphilis or alcohol is alone responsible, it is still more desirable to eliminate one or other from the etiology. For the purposes of differential diagnosis specific treatment is here valuable.

A diagnosis from *disseminated sclerosis* would be found in the absence of rhythmical tremors of the head, of spastic paralysis, and of hyperalgesia, in alcoholic paralysis with nystagmus.

The discrimination of alcoholic multiple neuritis from various *special atrophic paralyses* is sometimes called for. There are acute forms of polyneuritis in which at their onset the symptoms may be erroneously attributed to acute special paralysis and even to acute ascending paralysis (Landry's). Acute special paralysis usually shows itself quickly. So do some cases of acute alcoholic polyneuritis, mostly induced by a chill, though occasionally by muscle-tire. The rapidity of the onset is not generally equal, the alcoholic neuritis appearing more imperceptibly and slowly; but in these forms of alcoholic acute neuritis it is practically equal, the absence of sensory symptoms in the non-alcoholic paralysis and their presence in the alcoholic probably is the clearest guide to a correct diagnosis in the earlier stages of both diseases, there always being some pain, however little, in the alcoholic malady, but none in the non-alcoholic. The other early principal symptoms are common to both ailments, viz.: a flaccid paralysis of the limbs with loss of knee-jerk and the "reaction of degeneration." The course of the symptoms in a week or less discriminates the affection. In acute alcoholic neuritis the paralysis gradually extends, in the non-alcoholic acute spinal paralysis the paralysis reaches its full extent at once. In the former the affected parts only very slowly regain their power, in the latter the area of the paralysis may begin to contract gradually in from three to six days. In the acute ascending paralysis of Landry the paralysis ascends from below upward, beginning in the lower extremities, then affecting the trunk, and thence extending to the upper extremities; in the alcoholic neuritis the legs are affected first, then the arms, then the trunk, if the trunk be involved at all. In the alcoholic forms there are hyperæsthesia and myalgic pains and wrist-drop, if not foot-drop, these symptoms being absent in the non-alcoholic form. In the earlier stages when the differentiation of the diseases is difficult, the absence of pain and the continued presence of the knee-jerk point to Landry's disease. As the disease advances, the difficulty is lessened, but there are cases even then where the diagnosis is anything but easy. The muscular atrophy and electrical reaction of degeneration of alcohol, with their absence in acute ascending paralysis, will afford the greatest aid.

There is a close resemblance between the atrophy of alcoholic paralysis and the wasting of *progressive muscular atrophy*, but in the latter the alcoholic sensory disturbances and reaction of degeneration are wanting. Again, the progressive muscular atrophy producing the "*main en griffe*" commences in the hands, and not in the extensors as is generally the case with alcohol.

The absence of sensory symptoms marks an important distinctive symptomatic difference between general spinal paralysis (*chronic anterior poliomyelitis*) and alcoholic neuritis. Acute anterior poliomyelitis can at times be discriminated from peripheral neuritis only with extreme difficulty. The following distinguishing points are given by Buzzard: (*a*) Paralysis is more complete, and sensory symptoms are absent in acute anterior pyomyelitis. (*b*) Groups of muscles are attacked simultaneously in acute anterior poliomyelitis. (*c*) In peripheral neuritis there is tenderness on pressure over nerves.

In the case of certain *inflammatory diseases of the spinal cord* Pearce enumerates some diagnostic signs. In chronic meningo-myelitis there may be great wasting and severe pains, but the latter precede the paralysis. The tendon reflexes are increased, and there are other symptoms of spasmodic paralysis in the lower extremities. In spinal leptomeningitis there is deep-seated pain in the back, probably with spinal rigidity, and spasm of the muscles of the limbs. Cervical pachymeningitis begins with paralysis of the arms, not limited to the distal ends of the nerves, and there is a different history. In paralysis from pressure on the cord there is more or less paralysis of the bladder, a tendency to spastic paralysis, and to acute bedsores. Hemorrhage into the cord has a sudden onset, with paralysis of the bladder and sphincters. In diagnosing alcoholic paralysis, hysterical reflex and functional paraplegia must be taken into account. In alcoholic multiple neuritis the presence of cardiac disease and Bright's disease may be suspected from acute dilatation of the heart (the heart is a muscle which expands and contracts under certain conditions and is very dilatable), a weak rapid pulse, and the prominent œdematous (sometimes violet-tinged) state of the feet. The absence of albumin in the urine and of pulse tension will exclude Bright's disease, and the effect of alcohol in dilating the heart (as demonstrated by Richardson on a bullock's heart) and other phenomena of alcoholism will account for the condition of that organ.

Among other toxic acute paretic affections due to alcohol there is a variety, most baffling at first, which has been called *toxic hysterical paralysis*. It is characterized by a more or less sudden paralysis of some or all of the limbs. It is associated with a total loss of muscular sense, surface and deep anæsthesia, the regions implicated though clearly definable having apparently no relation to nerve distribution; flaccid paralysis of the limbs, or spasm without increase of the deep reflexes; loss of special senses on the affected side, color blindness, and restricted field of vision; no muscular innutrition; no facial paralysis

(Pearce). Hysterical paralysis also arises from non-alcoholic nerve disturbances and from poisoning by bisulphide of carbon and lead, and injuries, as well as alcohol. Charcot and Delpech think that alcohol, lead, and carbon bisulphide, besides producing special characteristic paralysis, may each give rise to the hysterical form. The onset on the whole is sudden, and recovery, so soon as the hysterical character of the affection is determined and suitable treatment applied, is rapid. There are a few distinctive symptoms, such as the dragging of the paralyzed limb, it being quite flaccid, no effort being made to raise it from the ground. Circumduction, which is present in organic hemiplegia, is absent as a rule in hysterical paralysis. The limb affected though pendent is not rigid, except the fingers, which are extended together and flexed at the carpo-phalangeal joints, making a right angle with the palm. Spasm in the fingers is shown by the recoil when they are bent forcibly, differently from the claw-hand of muscular atrophy. The arm and leg reflexes are not increased.

The diagnosis from *cerebral hemiplegia* is in great part founded on the fact that hemianæsthesia is very rarely seen in that disease, hemorrhage and degeneration seldom occurring in the posterior part of the internal capsule. In the few cases where hemianæsthesia and hemiplegia are of cerebral origin, facial paralysis of the side affected would point to the organic involvement, unless imitative facial paralysis (pseudo-facial paralysis of Charcot) is caused by glosso-labial spasm affecting the lower facial muscles. In these spasms when severe the patient cannot protrude the tongue; in the less severe cases, the tongue is protruded toward the affected side and is curved; in paralysis the tongue is protruded but not curved. Complete anæsthesia on one side, passing to the muscles and articulations, with loss of common sensation and muscular sense, insensibility to heat and cold, and analgesia, indicate an entirely hysterical origin, particularly when associated with loss of special sense, restricted field of vision, and color-blindness. The paralysis and contractions are on the same side as the anæsthesia in alcoholic hysterical paralysis. In hysteria the patient is usually unconscious of the anæsthesia. It should be remembered that spinal hemianæsthesia differs from that of cerebral origin, in there being no facial paralysis, and the anæsthesia affecting the side opposite to the paralysis. The prognosis is highly favorable if the hysterical origin of the symptoms is once ascertained. There are refractory cases, but these mostly occur in individuals who are either of highly neurotic temperament or have been addicted to alcoholism, in whom renewed attacks are specially likely to take place. The treatment is the more successful the more thoroughly the patient is under the physician's care. Though such patients generally do best in a hospital where they are completely under discipline, yet with an efficient and intelligent nurse recovery may always be looked for. Alcoholic intoxicant liquor should be rigorously discontinued, the diet should be chiefly milk, but any milky food or nourishing soup, or albuminoid meat extract, might be added as soon as the patient can swallow these. Rectal feeding is rarely needed, but forcible feeding may be necessary. While the emunctories require attention, the chief therapeutic reliance should be placed on antispasmodics with alteratives and tonics. Asafœtida with ammonia, calumba, or nux vomica with or without potassium iodide or bromide, or sodium iodide or bromide, all in small frequently repeated doses (every two or three hours), and strychnine are of great value. With all this strict injunctions as to hygienic habits, with as much exercise in the fresh air as possible, are generally required. To prevent recurrence of the spasmodic attacks abstinence from intoxicants should be enjoined, with simple wholesome food, eaten slowly, including wheat or oatmeal.

Besides all these diseases the toxic influence of alcohol on the nervous system shows itself by a great variety of *nervous affections* of a *mixed character*, in many cases exhibiting some of the symptoms of the different maladies which have just been considered, in such a commingling and in such degree as to defy classification. Polyneuritis accounts for only a minority of alcoholic nervous disorders. In 120 hospital patients treated for nervous troubles due to alcohol, Freyham (1894) found only 19 who could be classed as suffering from peripheral multiple neuritis of some kind. Of these the motor form was the most common, the ataxic coming second. In one instance a case of hemianæsthesia was put down to hysteria, aggravated by alcohol. According to his observation, the victims of alcoholism nearly all show great irritability and weakness in the ramifications of the central nervous system, constituting a morbid condition which may appropriately be called alcoholic *neurasthenia*, a grave unhealthful state which when present tends to intensify the unfavorable aspect of the prognosis. In the subjects of alcoholic neurasthenia the muscular irritability is extreme, the slightest mechanical stimulus setting up contraction, twitching of the fibrils, and tremor, but as a rule there is neither the reaction of degeneration nor paralysis. In the sensitive sphere there is a varying degree of hyperæsthesia, with heightening of the patellar and skin reflexes. In a large proportion of such cases it is only with the greatest difficulty that all other contributory factors except alcohol can be excluded, and a transition to typical neuritis is seldom noticed. In the diagnosis, general or differential, of all acute or subacute alcoholic toxic affections, the concurrence of some latent lesion of organ or tissue not directly connected with alcoholism ought ever to be before one, as the underlying, not necessarily alcoholic, morbid malady may modify the usual symptoms of alcoholic neuritis or other toxic alcoholic disease or process. Nor should it be forgotten that such a latent non-alcoholic or not directly alcoholic morbid state may at any moment take on activity, and thus still further modify the symptoms which have been attributed to alcoholic poisoning.

The best general rule for the *treatment* of all acute alcoholic maladies is total withdrawal of the toxic agent, as in metallic toxicity. Thirty years of professional life have taught the writer that there is no safer rule in the whole range of therapeutics. In an apparent call for alcohol in such cases, other drugs or combinations of drugs can be employed as effectually without that reactionary disturbance after the administration of an alcoholic intoxicant which is so fraught with fatal risk to life in most forms of alcoholic poisoning. He has seen a good many cases with an apparently favorable prognosis lost in the secondary reaction following the employment of an intoxicating beverage as a therapeutic remedy, especially in the more acute forms of poisoning by alcohol, such as delirium tremens; he has also observed it in the occasional cerebral commotions of subacute and more chronic cases of paralysis.

Chronic Alcoholism.

The various acute and subacute morbid affections which have just been under review subside, under appropriate treatment, the acute in the majority of instances within from one to three weeks, though their complications may at times extend further than the longest of these periods. In the greater number of cases the system appears then to recover its tone and the erewhile sick person to emerge from convalescence uninjured. This, however, is not absolutely true. After even a single attack of delirium tremens or mania-a-potu, time is essential for the reparation and renovation of the physical damage wrought on brain and nerve tissue, just as certainly as it is needed for the repair and renewal of the more manifest physical damage inflicted, during a drunken paroxysm in either of these alcoholic affections, on bone or muscle. Though not to the unskilled eye so necessary in the less visibly injured nerve structure, the period requisite for the reconstruction of normal cerebral and nervous structure is all the longer than that needed in the rehabilitation of injured bony or muscular texture, from the former being more delicate than the latter. In the thorough re-establishment of brain and nerve health, a long time must elapse before the constitution of the affected individual can be exactly as it was just prior to the illness, and if there be a recurrence, the foundation of permanent central nervous instability or of permanent physical degradation is too often laid. In this way acute alcoholism may, often does, by a pathological process of evolution or more accurately speaking dissolution, develop through subacute into chronic alcoholism, a downward progress of degeneration frequently so slow as to escape observation either from within or from without. There has been an imperceptible though none the less real change, the unfortunate patient and his friends being rudely awakened to the fact that he has become the most miserable of beings, a chronic alcoholic. In this form of alcoholic toxicity the symptoms are not usually so acute, but they are more prolonged, convalescence is retarded, the tendency to recovery is much feebler, the toxic lesion taking on permanence and intensity. It has long been a matter of observation that the man or woman who has a paroxysm of intoxication for a day or two once a month, preserving his constitution entirely free from the toxic influence of alcohol in the abstaining intervals, does less injury to body and brain by alcoholic toxicity (though he may run more risk of disaster by accident while intoxicated) than the steady, free "moderate drinker" so called, who with his four or five daily doses keeps his whole system continuously under the toxic influence. "Soaking" or saturating the blood with the poison daily for a long series of years is greatly more detrimental, more toxic in the infliction of permanent damage to the human constitution, than a periodic drunken bout, if the interval of abstention from the toxic agent be prolonged enough to enable the system to get rid of the immediate and remote toxic injuries.

Besides the gradual evolution of acute into subacute and thence into chronic alcoholic poisoning, daily continuous consumption of intoxicating beverages (say three to five or six times a day), in quantities often far short of what would by most persons be recognized as intemperate, in a very large number of instances initiates and carries on a train of pathological tissue degradations to which, though the symptoms vary with the individual constitution, the organs, tissues, and functions mainly implicated, the term "chronic alcoholism," in other words chronic alcohol poisoning, may justly be applied. In the slow (and for a considerable time invisible) increasing penetration of structure and function by the poison and the morbid products and disturbances resulting therefrom, the toxic symptoms are at first and for a time completely masked by the pleasing and exhilarative glow over frame and spirit, not as yet followed by a depressive recoil of any moment, or by bodily discomfort beyond the ordinary gastric headache or stomach-ache which even abstainers occasionally feel. Though there is not the pain and distress of acute alcoholism, the whole system is, though slowly yet none the less but rather the more surely, undergoing an advancing alcoholization. There is also greater permanence of injurious influence and tissue degeneration. Nutritional impairment alone suffices to silently and steadily undermine organ, degrade structure, and retard the normal physiological processes, converting these by degrees into pathological processes. The stomach and digestive apparatus is continually irritated by the repeated ingestion of alcohol which also precipitates the pepsin of the gastric juice, giving rise to the pyrosis, gastralgia, eructation, and other depravities of digestion which contribute to make life a misery to the chronic dyspeptic with his hypochondriasis and melancholia. The liver is gradually deteriorated in structure, becoming enlarged and fatty or contracted and cirrhosed. The kidneys are so structurally impaired as to be unequal to the adequate performance of their essential eliminative work. The heart is riddled with degenerated fat, hypertrophied, weak, and flabby, with incapability of any but slight exertion by reason of palpitations, dyspnœa, painful stitches, and præcordial anguish. This vital organ, from the tremendous overtaxing to which it has long been subjected, four ounces of alcohol (in brandy) having been shown by Parkes and Wollowicz to have caused 12,960 additional pulsations in twenty-four hours (equivalent to a strong young soldier of twenty-six years lifting $15\frac{1}{2}$ tons daily one foot high), is shorn of the greater part of its normal strength and power. The muscular capacity is lessened, as it were inch by inch, the experiments of Richardson and others having clearly shown that alcohol is a powerful diminisher of muscle-power. The tongue is often parched and coated or barked, the breath offensive and foul. Frequently there is persistent and clamant thirst, nausea, flatulence, intense pain in the region of the stomach, with anorexia and loathing of all food. The cook not unseldom comes in for a reprimand for bad cooking when the diner's alcoholized perversion of taste is the real offender. Feelings of extreme coldness or heat, of a variety of pains, twitchings, stabbings, indefinable variations of uneasiness are experienced all over the frame, especially in the back, legs and feet, shoulders and arms. The aspect of the chronic alcoholic is usually well marked and distinctive. In beer and other malt-liquor drunkards the subject even at an early stage has a tendency to obesity, which, as the disease advances, becomes more prominent and increases to an enormous extent, till he acquires a bloated and inflated appearance, puffing and blowing with the slightest exertion. The features are heavy and dull, the face red and somewhat of a purply hue, especially on exertion, with vascular hypertrophy most conspicuous in the regions of the eyes and nose, with blotches and an oily, greasy, glistening surface, conjunctival yellowness (bilious or fatty), the eyes moist and red. In the early stages the beer drinker may be quick and active, but gradually his gait loses much of its elasticity, he looks more sluggish and embarrassed in his movements. Dropsy or syncope or embolism frequently closes the scene in middle life. Spirit drinkers as a rule are more shrunken in aspect, and often grow thinner the longer they continue their deep potations, till in many cases they are quite emaciated. From their liability to cirrhosis of the liver and kidneys and to cardiac fatty degeneration, they often display, as the disease advances, the abdominal rotundity of ascites as well as the œdematous girth, especially in the lower limbs, of anasarca. The brain and nervous system are the seat of much disturbance. Spasms of muscular irritability and restlessness are a common phenomenon from almost the first. These are more apt to occur in the early morning on awaking, particularly before food, a meal for a time allaying the tremors which, spreading from the legs upward, attack the upper limbs and then extend over the whole frame. Slowly and imperceptibly the control over these tremorial spasms and the power of food to calm the agitation diminish, till, after a longer or shorter period, the seizures are oftener repeated, and finally the tremors grow stronger and abide, vertigo and a sudden tendency to fall forward or backward, with almost momentary sensations of swimming in the head, with transient undefinable feelings of dull aches and heaviness principally of the head, though similar uncomfortable sensations are experienced throughout the body, worry and annoy the chronic alcoholic, which he by an effort of will at first controls, but soon loses all power to do so. Disturbed sleep, from which he awakes unrefreshed and weary, is an early symptom. Though formerly dreamless, the sleep insensibly becomes less sound, and appalling and horrid dreams deprive the sufferer of the proper rest which is so indispensable to recovery. Insomnia is a terror to chronic drunkards, especially to those cultured neurotics who are so susceptible to the action of narcotics. The too frequent practice of a spirituous "night-cap" in such cases, though it may induce sleep, induces a sleep which is only of a superficial kind, disturbed and uneasy generally, and if deep, neither refreshing nor restorative. The less usual but still frequent habit of taking an opiate or other narcotic sleeping draught is never free from risk, and the premature death of not a few accomplished literary men and women as well as of members of the learned professions has had this pernicious and dangerous practice for its cause. The special senses are subject to disorder, such as various kinds of noises in the ears, muscæ volitantes and appearances of figures, lines, or flying things or other forms, sometimes weird, floating before or within the eyes, even when the latter are closed, though at first closure of the eyelids puts them to flight. These illusions, with auditory disturbances, at times combine to engender delusions of persecution, the sufferer from which is almost continually, when awake, harboring the belief that the ghost of some one whom he fancies he has injured haunts him, or that some living person is ever on his heels, for some imaginary violent purpose. One inebriate married lady between fifty and sixty years of age, a chronic alcoholic, has again and again rushed in terror into the writer's house or sought protection from a constable, to save her from being (as she affrightedly declared) indecently assaulted by an old lover of hers more than thirty years before, who she thought was just behind her. In a few cases of this distressing persecution-mania a single delusion will persist to the end, or another may after a time take its place, for a term of months or years; but the writer has never seen two different delusions of persecution coexist in chronic alcoholism, except during an intercurrent attack of delirium tremens or acute insanity. If the victim does not recover, after hurrying from one town or country to another, he may terminate the torture by suicide in despair of getting rid of his tormentor. Double consciousness is a striking feature in some chronic alcoholics. So real does the double state feel that the patient can (when just awake) find himself as it were coming slowly back to himself from his other self, and he can sometimes remember what he was doing or thinking in that other condition. At other times he can remember only partially. There is a second form of double consciousness, when the subject can feel that he is two separate persons at the same time, and talks or sings to his other self just as the separate heads and upper halves of the "Two-headed Nightingale" conversed with each other. This morbid brain condition is grave, but the writer has known it to be set right by treatment directed to the brain, with immediate discontinuance of the toxic agent. This double consciousness must not be confounded with the hallucinatory belief of the presence of another person when no one else is present. In the latter case the alcoholic talks to an imaginary some one else, in the former he talks to himself in an imaginary second existence. All through a career of chronic alcoholism runs a thread of mental degradation involving moral obliquity. Indecision of character, loss of thought-concentration, vague fears of impending disaster, irritability and violence of temper, uncertainty and infirmity of purpose, are accompanied, apart from other evidences of depraved morality, by a specially characteristic trait of falsehood. Mendacity is almost a diagnostic mark of chronic alcoholism. Lying becomes second nature. Not that the writer believes that such inebriates lie of design voluntarily. They cannot help it. Partly because perception is blurred they do not see things as they are in reality, but only as seen in a clouded mental atmosphere, where everything assumes a hazy, contorted appearance, the poisoned brain all the while having by physical degradation produced mental and moral palsy. The writer has known inebriate ladies of culture and excellent principles solemnly declare that they had not been drinking while the aroma of the just-drunk brandy was already being diffused with their every word, and the tell-tale tumbler had just been laid down. Even on their death-bed from alcoholic poisoning he has heard similar untruthful denials.

The issue of all these bodily and cerebral disorders is often the grave condition of alcoholic dementia, and the premature ageing of the poisoned subject. The antedating of mental decay by from five to twenty years may be seen in many persons who have had no direct or indirect alcoholic inheritance, as the effect of chronic alcohol poisoning, as well as in constitutions born with an alcoholic taint. Sometimes this fatuous demential state is unaccompanied by visceral disorders, the brain and nervous system having borne the brunt of the alcoholic toxication. While the chronic alcoholic is a dement, vacant, silly, and foolish, he is usually tremulous and somewhat ataxic. The sharp pains are so mainly located in the upper and lower extremities as to have given rise to their being known as alcoholic arm and leg pains. The tongue is tremblingly protruded and as the malady progresses the chronic alcoholic becomes the subject of increasing and general trembling of the muscles, which often assumes the appearance of paralysis agitans. Occasionally there is hemianæsthesia with sensory impairment on the affected side, which is lower in temperature than the side which is not involved.

The diagnosis from *general paralysis* in certain stages is by no means at first sight easy unless one has an accurate history of drinking. The matutinal nausea and emesis, foul breath, furred tongue, eructations, gustatory depravity, and gastric distress, point to an alcoholic factor, as do the anorexia, headache, anæsthesia, tremors, and diarrhœa. Constipation is common in paralysis. Nervous excitement, unrest, uneasiness, fear, wakefulness, and other symptoms of delirium are common to both diseases, but are more pronounced in alcoholism. Further alcoholic diagnostic marks are the bloated and puffy, pale and waxy complexion, shuffling ataxic gait, listlessness and dulness of expression, the impaired or perverted sensation, and occasionally amaurosis (there is also a nicotine amaurosis), with usually but not always the characteristic odor of the breath. Many forms of motor and sensory paralysis are caused by alcohol besides those already described under the acute form. The most common is alcoholic paraplegia which may persist for years, though the writer has seen recovery in most cases where alcohol has been renounced. When it goes on, this paraplegia generally ends in dementia. General paralysis is also induced by alcohol, and is discriminated from the non-alcoholic form by being antedated. In the writer's experience the antedating has been from five to thirty years, and the proportion of cases of general paralysis, in which alcohol has been a leading factor, has been twenty-two per cent.

Pathology and Post-Mortem Appearances of Acute Alcoholism.

A sufficiently large dose of alcohol in a strong alcoholic liquor, whiskey, gin, brandy, or rum, acts as a narcotic poison on the nerve centres, and causes death by coma, sometimes very rapidly. Many such rapid deaths have been recorded. In such sudden cases a large quantity of some form of ardent spirit has generally been swallowed within a few hours, while little or no food was being taken. In one case of the writer's, a workingman, aged thirty-eight, who had been drinking whiskey without eating for four hours, fell down dead at the bar of a public house. In this case a post-mortem examination disclosed one of the modes of sudden death sometimes occurring after rapid and heavy drinking. The heart had given way, there being a rent in the wall of the right ventricle half an inch long, and the pericardium was distended with coagulated blood which had poured through the ruptured organ. In another case, a scene-shifter, aged forty-one, dropped down dead on a theatre stage behind the scenes, on a pantomime night. The stomach was distended with a mass of undigested food, the gastric mucous membrane angry, inflamed, and eroded in patches with grumous exudation, the lungs exhibited extravasations of blood, the meningeal membranes were congested with subarachnoid effusion, the heart, the walls of which were pierced with fatty degeneration, was pale and flabby. The right ventricle was distended with a large quantity of semifluid blood. In this case the deceased, who had been previously in apparent good health, had been drinking heavily and frequently of whiskey and brandy for some sixty hours. In a third case of a woman, aged forty-three, who had been intoxicated for some three days, death took place suddenly from the rupture of a blood-vessel in the brain. At the same time it should not be left out of account that, in certain states of ill-health or exhaustion from insufficient food, prolonged loss of sleep or exposure to extremes of heat or cold, of nervous excitement or asthenic disease, a quickly fatal termination may follow from smaller quantities of spirit or even of less potent alcoholic beverages. Again, alcoholic insensibility may supervene in a few minutes, or not for hours and then suddenly without any warning. Further, a person may to all appearance recover from the confused thought, giddiness, tottering gait, inability to walk, and stuperose state of deep intoxication, with or without free vomiting, and after a time without any premonitory symptoms become unconscious and die convulsed. Intoxication with complete recovery is a lesser degree of alcoholic poisoning, comprising the exhilaration stage of vascular relaxation, cerebral automatism with incomplete paralysis and muscular inco-ordination, and lastly the advanced paralysis, unconsciousness, and automatic existence of dead drunkenness; all these phenomena constituting a drama of three acts of progressive paralysis. Intoxication is sometimes accompanied by acute congestion of the alimentary canal, especially of the stomach and duodenum. When death by accident occurs during acute intoxication, the brain and its membranes are seen to be congested. This has been observed by Richardson in a man who, when intoxicated, fell off a railway platform, and was struck by an engine in motion, the accident laying his brain open. What the writer has frequently seen in a post-mortem examination of persons who had died when drunk, apart from chronic changes, has been general hyperæmia, most pronounced in the cerebrum and meninges; gastric vascular injection, the injected red-looking vessels presenting the form of a tree with branches, a seeming *arbor mortis,* the injection extending into the duodenal vessels. Sometimes in the brain and on its surface there were small extravasations of blood. The capillaries in several cases were distended, with consequent engorgement of the cerebral venous system. Renal hyperæmia was common, and in a few cases there was a hobnail or cirrhosed liver. The latter form the writer has seen only in 1¼ per cent. of several hundred post-mortems of both acute and chronic alcoholism. The most frequent form met with was the nutmeg and fatty. In many cases there were evidences of former pleural effusion (adhesion to the parietal pleura), and in about 23 per cent. signs of old pericarditis (white spot on the heart).

Post-Mortem Appearances of Chronic Alcoholism.

In the bodies of inebriates of long standing the tissue changes were more distinct and permanent. The stomach usually presented a darker bluish-red hue, in striking contrast to the general blanched appearance of the mucous membrane, with patches of ulceration and erosion. Thinning of the muscular coat was common, so that when the stomach walls were stretched transmitted light revealed a semi-transparency. There was usually an increase of the submucous connective tissue with atrophy of the gland cells from chronic gastric congestion and catarrh. Dilatation of the stomach was noted mostly in free drinkers of beer or porter. With shrunken cirrhosed, or nutmeg or fatty liver, the spleen was often enlarged. Evidences of old pleuritic adhesions, lobar emphysema, bronchitis, and hypostatic pneumonia were frequently found. The definition of kidney structure was generally barely perceptible. The brain was less hyperæmic than in acute cases of intoxication. Only in a few bodies were general cerebral congested bleeding points found. In two cases of the sudden death of chronic inebriates there was rupture of an aneurism of the middle cerebral artery, while in a number of cases death was caused by rupture of some other cerebral vessel. Arterial dilatation was present in a large proportion of cases, with atheromatous thickening of the coats of the arteries, accompanied with tortuous vessels, the smaller in calibre being coarse and tough; there were sometimes atheromatous deposits on the endocardium, and the veins were more or less varicose. These degenerative changes extended considerably through the general vascular system, diminishing the calibre of the vessels. The heart was often loaded with superincumbent fat, flabby, pale, infiltrated with or degenerated by fat, dilated or hypertrophied, the latter probably compensatory to asthma or fatty granular renal disease. As a rule spirit-drunkards show a greater increase proportionally of connective tissue, this accounting for their greater emaciation, hollow cheeks, sharpness of feature, aged, withered and puckered face; while malt liquor and wine intemperates have a deposit of fatty tissue under the skin and in the viscera, as well as in the heart and generally throughout the body, to some extent taking the place of bony tissue in the framework, the individuals being corpulent, with greasy, oily, glistening skin and abdominal protuberant fat even when there is wasting of the legs and arms.

In the brain there is generally a shrinking of the mass of substance with narrow, flattened, and shrunken convolutions, which appear to be more clearly defined, while there is often serous effusion in the ventricles and in the subarachnoid space. From the degeneration of the vessels there is sometimes found blood which has oozed into the brain substance. There is a general wasting of the nerve cells and nerve fibres, with a similar atophy of all the nerve centres which are somewhat tough. The nerve cells very frequently are altered in shape and capacity by the great proportional increase of the connective tissue. This neuroglian proliferation is a prominent feature. The dura mater is often found to be abnormally adherent to the cranium, the Pacchionian bodies enlarged, with opacity of the arachnoid and thickening of the pia mater. On the surface of the dura mater there is sometimes neoplastic cellular exudation, vascular and at times hemorrhagic, indicative of pachymeningitis. The pia mater is generally opaque, thick and coarse, adhering to the brain surface with degenerative patches. Arachnoidal hemorrhage with excess of serum is also met with. Bevan Lewis ("Textbook of Mental Diseases") gives a masterly description of the pathological changes found in the brain and membranes. The vessels dipping into the cortex from the pia are enlarged, coarse, often tortuous, with their coats in advanced atheromatous and fatty change. The numerous adventitial sheath nuclei are freely proliferating, or their protoplasm is undergoing fatty degeneration. The most prominent feature is the profusion of scavenger cells, pervading the upper or outermost region of the peripheral zone of the cortex lying immediately behind the pia, their branching processes forming a dense matting which converts the outermost fourth into a closely felted substance of minute meshes. These scavenger cells are most numerous where a blood-vessel passes downward through the cortical layers, dipping down into the second layer, in aspect like the connective-tissue increase along Glisson's capsule in hepatic sclerosis. This matting is denser just underneath the pia. In the epicerebral space between the pia and the cortical surface amyloid bodies are seen. Congeries of proliferating nuclei are seen here and there in the vascular walls, fibrous extensions from which penetrate the perivascular cortex, the vessels thereby presenting a spinous aspect. The deepest cortical layers are those specially affected, the upper above the fifth being sometimes apparently not invaded. This morbid evolution is initiated in the vascular elements, in the long, straight cortical vessels. The motor area is that chiefly involved. In the lowest layer—the spindle-cell formation—scavenger cells suddenly present themselves. Nuclear proliferations often conceal the spindle-cells from view, the nerve elements being preyed upon by the scavengers. The vessels are the seat of atheromatous and fatty changes, the walls being covered with young spider-cells. In the spinal cord there is increased vascularity, or perhaps more prominent obtrusion of vessels, the anterior columns being least involved, the posterior median raphe and columns being usually those most affected. In some cases there is thickening of the muscularis which may encroach on the cavity of the vessel itself, the non-elastic intima being in consequence made to assume a plaited form. Sometimes the vessel is occluded by this muscular tissue. This change is practically identical with the increase of the muscularis discovered by Sir George Johnson in chronic Bright's disease. In many cases this has not been observed, owing to less concentration on the vascular supply of the cord than of the brain.

The appearances after death from *alcoholic progressive multiple neuritis* had till recently disclosed as a rule no spinal lesion except a softening of certain portions of the cord by Clarke (*Practitioner*, December, 1881). But Eichhorst found a few diseased patches in the mid-dorsal region, disease of the small vessels throughout, with increase of connective tissue in the lateral column. Inflammatory changes and degeneration in some ganglion cells were found by Schäfer, Payne, and Sharkey. Pal noted degeneration of Lissauer's posterior root zone in the lumbar region and general involvement of Goll's columns; and, in another case, intense degeneration of Goll's columns in the cervical region, less marked in the dorsal, but appearing again in the lumbar segments. In both cases there was disease of the nerve roots. Thomsen has seen disease of the nuclei of some of the cranial nerves in the pons and medulla oblongata, while Hun and Kojewnikoff have found slight degenerative changes in the ganglion cells of the cortex cerebri. Déjerine and Sharkey have described disease in the vagi and phrenic nerves. These appearances have been observed also by Campbell (Transactions of the Pathological Section of the Medical Institute of Liverpool, February 23d, 1893). The seat of the disease, however, has been found to be chiefly peripheral and generally limited to the finer nerve terminations; the intensity of the morbid process, where the degeneration has extended to larger nerve branches, diminishing in proportion to the distance from the periphery. The morbid changes are nearly if not always symmetrical in both lower and upper extremities; the former being the more frequently affected. This peculiar inflammatory degeneration of the peripheral nerve fibres may be parenchymatous (in the nerve fibres themselves, or interstitial, involving the tissue in the interstices), or external (in the sheath), all occurring more or less in each case. On the whole most of the inflammatory changes have been found to be parenchymatous, but as to whether these are primary or secondary has been a matter of controversy, some attributing the degeneration to strangulation of the nerve fibres higher up. The degeneration which has sometimes appeared in the vagus is credited by some with the acceleration of the pulse rate which has characterized certain alcoholics. By other observers traces of congestion of the pia mater have been reported. Sometimes there is inflammatory swelling of the whole affected part, but frequently there is no perceptible lesion except on microscopic examination, when the nerves may have a dull look and have lost their normal shape from fatty myeline degeneration. This degenerated portion, at first cloudy, gradually separates into segments which alter till they surround the axis-cylinder as fatty particles which increase and develop into granulation corpuscles, obliterating wholly or partially the axis cylinder. The changes in the sheath and interstitial tissues consist largely of an enormous increase of the nuclei of the sheaths and infiltration with leucocytes, with thickening of the perineurium. Finlay also records wasting of the fibres of the extensors of the wrist, the interstitial spaces being crowded with leucocytes and nuclei. While in peripheral forms of alcoholic paralysis the morbid appearances are mainly peripheral, in alcoholic dementia and insanity they are central. The latter appearances are brain shrinkage and softening, interconvolutional shallowing of the furrows, tortuous atheromatous vessels, and ventricular effusion of bloody serum.

Pathology of Drunken Acts.

Though to the non-medical spectator an intoxicated person appears to be simply a man beyond his own control and more or less bereft of reason and judgment, the intelligent physician discerns something more and deeper, the pathological effect of a narcotic anæsthetic on the brain. To the skilled scientific observer the phenomena of drunkenness, the mental paresis and the inco-ordinate gait, are as plainly indicative of brain-tissue deterioration by a poison to the intelligence (when other mentally disturbing agencies are not present) as are the blue-green line and the agonizing *colica pictonum* of the irritant poison lead. Intoxication is a state of brain disorder, the manifestation of a poisonous action on brain tissue and function, arising from the alcoholic supersaturation of brain and nerve tissue, and of the blood supply of the brain and nerve centres. These pathological acts of drunkenness, each a manifestation of renewed alcoholic poisoning, in the ratio of their recurrence deepen and confirm the effects of the preceding acts, unless after each act there has been an interval of abstaining life long enough to allow the system to recover its physical integrity. This non-alcoholic interval to be effectual must after a first poisoning last for at least three months, but even then the poisoning of the nervous fluid will not have been absolutely recovered from. The mental instability after a single bout of intoxication has been known to continue for nearly a year. The coarse lesions such as the gastric distress, the congested brain, the disordered liver, and the irritated kidneys, however, are soon under abstinence a thing of the past; but these organs and tissues, when alcoholically upset with increasing frequency, soon begin to be acted on by a continuous chain of poisoning, which first inflames and irritates, then impairs organic and functional vigor, and finally destroys capacity with structure. Apart from acts of intoxication, alcohol regularly and freely ingested, even when taken daily in no larger quantities than one and a half ounces for adult males and one ounce for adult females, exerts a poisonous influence. Alcohol alters the chemical composition of the blood, diminishes its capacity of oxygen-absorption, lessens its corpuscular vitality, and impairs the nutritional processes. This impairment of nutrition is increased by the diminished calibre of the blood-vessels, an effect of several causes, the chief of which is alcoholic paresis of the vaso-motor nerves, aided by fatty deposits in the coats, followed by atheromatous and allied degeneration; though the enfeeblement of the heart itself by alcoholic overtaxing and physical tissue-degradation, sensibly adds to the mischief. Alcohol thus causes nutritional inadequacy by its morbific action on both the blood and the vessel walls, inclusive of those of the heart. The blood is frequently still further devitalized by alcoholic interference with the elimination of the waste products, the undue retention of which renders this fluid less potent in the construction of sound tissue. The texture of various organs is riddled with fatty degeneration so that their depurative constructive and eliminative capacity is also weakened. A characteristic abnormal effect of alcohol is its lowering the temperature of the body, and thus depriving the system of some of the vital heat so essential to healthy life. All this imperfection and perversion of the nutritional process by continuous operation tend to produce molecular changes which develop into permanent pathological states of cell degradation. This physical degradation not only affects the brain condition of the living, but has a tendency to reproduce itself, modified by the condition of the mother and other influences, in the offspring. Further, when this transmission is due in the first place to alcoholic physical degradation, it is much oftener met with than a similar transmission originating from any other cause. Thus morphinism is not nearly so often transmitted. The peculiar affinity of alcohol for brain and nerve substance and the specially marked tissue degeneration of alcohol are probably the explanation. This peculiarly degenerative influence on cell life may also be the cause of the greater susceptibility of certain persons to the narcotic capacity of alcohol. This susceptibility, which renders it impossible for such individuals to taste an alcoholic beverage without either showing signs of alcoholic disturbance or experiencing an overpowering impulse to drink to drunkenness, is more delicate when from an alcoholic than from a non-alcoholic source. The morbid drink impulse, too, which may arise from various disordered brain and nerve conditions, is more intense when the result of alcoholic physical degradation. This impulse involves the highest nerve centres, physically affecting the most delicate intellectual processes, as it dims perception, blunts sensation, and reduces muscular power. The integrity of inhibition is indispensable to sound mental, moral, and physical health. Alcohol, by its vitiation of the blood and its narcotic action on the nervous fluid and the intelligence centres, is a potent factor in the genesis of acquired, as distinct from inherited, deficiency of self-control. By its specific influence on the circulatory and nervous systems, its tendency is to impair the inhibitory function. The first stage of alcoholic action, when the circulation is quickened, the spirits are raised, and the face is flushed, is one of paralysis. The vaso-motor nerves have been paralyzed, their capacity has been reduced, their vigilance relaxed. From the lessening of the check on the heart by the paralysis of the cardiac inhibitory centre the pulse becomes bounding; from the loss of power in the higher inhibitory centres the affected individual is at once garrulous, confiding, pliable, and elated. Each renewed inebriate act still further reduces inhibitory capacity. The stock of inhibition, central and local, in many cases from various causes by no means over-abundant to start with, is by long-continued withdrawal, through the daily consumption of intoxicants, gradually if insensibly decreased to the utter loss of control. Alcohol is the most powerful waster of inhibitory force, in the higher nerve centres so surely weakening inhibition as, in numerous persons, so practically to obliterate the faculty of self-control as to leave the subject powerless to resist the slightest temptation to intoxication. By its progressive paralysis of inhibition of the local nerve-centres their capacity is so impaired as to undermine the vigor of organ and tissue, whereby morbid functional disturbances arise which operate as influences exciting to drunkenness, the alcohol all the while reducing the resisting and controlling power. Healthiness of function is correspondent to healthiness of cell life, alike in body and brain. The latter organ, from the greater delicacy of its structure, is the more quickly responsive to, and the more intensely affected by, alcoholic disturbance. As alcohol impairs cellular vitality, it thus by direct action on brain and nerve tissue sets up pathological states of cerebral and nervine disorder. Alcohol, being as subtle and far-reaching as it is potent in its poisonous narcotic action on an organ with the extreme delicacy of brain texture and mental processes, has a specially noxious influence on the brain, which injurious influence is intensified by the keen affinity of alcohol for brain and nerve element. By its physical tissue-degradation alcohol poisons brain and nerve, while by its narcotic property it depraves and impairs brain and nerve function. Alcoholic inebriety is therefore a central disease of the nervous system, a dominating neurotic malady beside which the liver and other visceral alcoholic organic lesions with those of the thoracic organs, grave as they are, are of comparatively slight significance.

The *modus operandi* of alcoholic injury of the brain, through the circulation, is worth notice. Alcohol is conveyed by the blood to the meningeal enfolding envelope of the brain. By paralysis of the vaso-motor nerves control over the vessels is relaxed, causing their dilatation with an increased supply of blood to the brain. The result is temporary functional cerebral excitement. This is the period of exhilaration and elation. The vascular contractility having been reduced by the alcoholic vaso-motor paresis, the increased blood supply cannot be sent back from the brain rapidly enough, and a block of the circulation occurs which impedes a renewed supply of freshly aërated blood. This is the period of depression, weariness, and dulness. If within a certain time after the termination of this pathological process the acute toxicity be frequently repeated, permanent tissue changes are the result. The membranous envelope is thickened and altered so that the brain does not receive a good and sufficient nutrition supply. Thus the brain is badly nourished. The alcoholic dense matty connective tissue by hyperplastic aggregation intrudes upon and obliterates the brain cells or alters their form, so as to impoverish them as well as lessen their functional capacity. This physical destruction and degradation of meningeal and cerebral substance cannot but impair the intellectual and moral faculties. Perception is blurred, the intelligence clouded, sensation dulled, the disposition perverted, will-power enfeebled, and the moral sense blunted. These changed and depraved faculties and functions are often directly traceable to brain-tissue degeneration produced by slow alcoholic poisoning.

As the brain and intellect are depraved in function by the morbid tissue changes produced by alcohol, so is the moral sense. By its anæsthetic influence alcohol takes the fine edge off perception, so that the dulled perceptive faculty cannot see or feel the physical and psychological damage done to texture of body and impairment of intelligence. In this state of partial anæsthesia the drinker neither feels nor suspects that anything is amiss within. He is therefore often utterly unconscious of danger, even when it is evident to all around him that he is drinking himself into his grave. While his senses are benumbed under this state of narco-anæsthesia he feels no ache, when if he were not to some extent anæsthetized he would be groaning from physical pain. Nor is he alive to the diseased undermining of his vital organs, or the deterioration of his frame and muscular power. He is borne headlong on his career of chronic alcoholism either blind to the dangers which are before him or, if he sees them at all, beholds them only as in a confused, cloudy, indefinable mist which makes no impression on his mind and conveys no sense of peril to him. This narcotic paresis involves all mentalization. He can neither comprehend to the full, nor discriminate accurately. He cannot measure the amount of force which he puts forth any more than he can calculate approximately the amount which is required for the accomplishment of any effort. Thus he may quite unknowingly and without any intention to do violence be guilty of manslaughter. Not only when intoxicated but when laboring under the anæsthetic influence of regular and continued free alcoholic drinking, his mental acumen may be so enfeebled that he may strike a blow violent enough to kill one, when he intended only to give a slight push. This paralytic action of alcoholism may involve all the faculties, so that he can have only faint and confused perception of facts, appearances, and morals. The mendacity of the chronic drunkard is proverbial, yet he is not necessarily an intentional liar. As he does not see things as they actually exist but only as it were through tinted mental spectacles, so his feelings are often the mere deceptive sensations of an alcoholically disordered brain. The inefficient mentality and the perverted moral sense of the chronic alcoholic not unseldom lead to unusually immoral and indecent deeds, as the alcoholic poison gradually and steadily extends and deepens its malign influence over the whole man. These acts of indecency are often the effects of physical tissue changes in the cerebrum and higher nerve-centres, though the acts may not be done till long after the person may have given up the drinking of alcoholic intoxicants. As in the post-paroxysmal intervals of epilepsy and the post-paroxysmal periods of periodical inebriety, when maniacal violence may suddenly occur, so in chronic alcohol poisoning, the now abstaining though erewhile intemperate subject may be a prey to post-inebriate indecencies and other aberrant symptoms resulting from tissue degeneration initiated by alcohol years previously. This moral declension is partly due to the tissue-depraving influence of secondary poisons (secondary to the alcohol) arising from the undue retention of waste products, the elimination of which is retarded by alcoholic interference. By its direct poisoning action, by its lessening and vitiation of the blood supply to the brain and nervous centres, and by the secondary excessive retention of carbonic acid, urea, and other poisons, a chain of pathological changes is set up, which, while disturbing brain and nerve function and antedating senile decay, enfeebles, distorts, and degrades the moral sense. This series of abnormal retrograde alterations, all of them practically symptoms of cerebral mental and moral dissolution, is as really a progressive physically initiated paralysis of mind and morals as is hemiplegia, or the dropped feet and hands of alcoholic peripheral neuritis or lead poisoning. The explanation of this alcoholic mental and moral paralysis is to be found largely in the alteration of nerve structure and function. To have the capacity to acquire accurate knowledge, consciousness must be healthy and complete. Being a complete act, each part must be accurately performed. If sensation be dulled by alcohol nervous structure cannot take on an accurate impression, nerves cannot transmit accurately. Even with only perception imperfect, consciousness cannot be wholly accurate. Still less accurate and complete must a conscious act be if representation, ideation, sensation, and volition are also defective. The staggering gait and incoherent speech of an intoxicated person are evidence of the partial paralysis of the muscular system. The deceit and falsehood (though the latter may be unintentional) of the alcoholic are indicative of a partial paralysis of the nerve centres which regulate the physical seat in the brain of our moral nature. Complete universal paralysis is death, but incomplete universal paralysis is disorganization of function. An inebriate whose whole nervous organism is thus partially paralyzed can neither reason, judge, nor discriminate in the elucidation of obscure and intricate problems. Alcohol, besides vitiating the blood, irritating tissue, and undermining vital organs, destroys nutritional co-ordination so that the relationship of various parts of the body is rendered asymmetrical. Fibro-cellular structure is specially subject to this diseased and inordinate growth. This morbid excessive increase of substance is so evident in such organs as the liver, kidneys, and brain, and so productive of structural and functional disorganization that, in the words of Sieveking, there is not a degenerative bodily condition which may not result from alcoholism. A sudden moral lapse, in an alcoholic of former high moral tone, is often as pathognomonic of an alcoholic lesion in the cerebral fibrous connective tissue, as is cirrhotic ascites of a like lesion of the liver. This abnormal fibrous-tissue proliferation by intrusion and pressure alters the shape of the nerve cells, some of which are strangled by the continued pressure followed by contraction of the reticulated matty fibrous structure. In this way nerve cells and nerve centres are by the lessened blood supply so starved as to waste and die. By and by the physical degradation of the central nervous tissue is assimilated to the system and modifies the constitution, which constitutional modifications can be transmitted to some extent. In addition to all these pathological degenerations of tissue, with the inhibitive intellectual and moral damage, alcohol enfeebles volition by a progressive paralysis of the will. A victim of chronic alcoholism may have a knowledge of his past and present state, may be so alive to each downward step to death, as to be plunged in remorse in his intermissions of sobriety, may even form the apparently strongest determination to drink no more; yet, with all his good resolutions, in spite of the most desperate efforts to abstain, he may be utterly powerless to refrain from intoxication. This volitional enfeeblement, this will-palsy, is frequently the product of the pathological progressive reduction of will-power which alcohol exerts in virtue of its being a potent will-paralyzant. The writer has shown how, by a lengthened chain of morbid degenerative changes ("Inebriety, its Etiology, Pathology, Treatment and Jurisprudence"), capillary paralysis, circulatory tumult, vascular atheroma, cellular decay, membranous thickening, cerebral congestion, and neuroglian proliferation, alcohol dims the perception, confuses the judgment, paralyzes the will, and deadens the conscience; and how deficient and perverted nutrition has been the initiation of this series of pathological, physical, intellectual, moral, and volitional degradations. But there is more. By irritating liver, kidneys, and stomach, alcohol persisted in causes pathological structural organic lesions which impair those depurative and digestive functions on the efficiency of which health so largely depends. The alcoholic depraving of the blood, derangement of function, and poisoning of the organism give rise to the morbid intoxication impulse and crave. Thus by pathological action alcoholic poisoning induces the disease of inebriety or narcomania. In no form of mental alienation are delusions of persecution more prominent than in alcoholic insanity. One does not find these delusions so frequent in unsoundness of mind from morphine. Why are delusions of persecution, and it may be added of poisoning, so often met with in alienation of alcoholic origin? The pathology of these and other delusions of suspicion gives the explanation when the action of alcohol on the heart and circulation, the liver and other organs, then on the brain and nerve centres and on nutrition, is taken into account. Alcohol accelerates the pulse, relaxes and dilates the heart and large vessels, causing heart failure with diminution of vascular elasticity and tone. The alcoholic fatty organic degenerations embarrass the breathing. The starvation of the brain and nerve centres induces cerebral anæmia and an irritable state of the nervous system, leading to brain and nerve functional depravity and perversion, with the issue of depression, uneasiness, and suspicion from these unsatisfied and therefore discontented mental wants.

The pathology of the tissue-crave deserves consideration. By partly perverting and partly impairing function throughout the system, by its affinity for water which causes it to freely take up that fluid, by its vitiation of the blood, by its interference with secretion and excretion, alcohol in many subjects so depraves the tissues that these, as soon as the anæsthesia of a dose has disappeared, pathologically cry out for a fresh supply of the anæsthetic. In such cases the whole system is in an agony of physical distress, so that, even though the patient loathe the taste of the liquor, he cannot resist the universal and intense tissue-crave for more anæsthetic relief.

Treatment.

The treatment should involve unconditional disuse of all alcoholic intoxicants. That in itself will alleviate most cases, and on that basis alone can any hope of cure be fairly entertained. The alcohol should be at once withheld, and there should be no compromise with the patient on this point. The writer has never seen any injury resulting from this "heroic step." "Tapering off" is inadmissible with alcohol, though generally the better course with opiates. It is judicious to give the patient something to take instead of the liquor, simply on account of the effect of long-continued habit which, though innocent, has a strong tendency to stamp an impression of itself on the brain so distinct as to cause that organ automatically to direct any action which has been frequently repeated. Hot water is better for this purpose than anything else, flavored or unflavored with a little lemon or lime juice, or cinnamon, or ginger. Cold water suits other chronic alcoholics better. Others are better served by coffee, weak tea, cocoa, milk, etc. In a few days the number of drinks can be rapidly diminished, these substitutionary draughts, not always required, being more for the temporary relief of discomfort than from imperative need. When such cases come under treatment there are frequently nausea, sickness, and other symptoms of gastric distress. Effervescent mixtures (or bismuth combinations), with milk and soda or lime water, are then indicated. As the sickness diminishes, concentrated albuminoid food, broths, and light farinaceous diet should be gradually supplied, followed by some variety of white fish such as sole, whiting, haddock, cod, or plaice, then white meats, and lastly a return to ordinary diet. Peptonized food is excellent at first in some cases. The period of gastric distress may last for a week or ten days, but this is exceptional, most patients having recovered their normal appetite within a week. There are cases in which the appetite has not failed. In these and in the majority of cases, after the cessation of the gastric troubles, good wholesome meals of meat, vegetables, and fruit are the most useful diet. The dietetic question is of high importance. It has been alleged by some that a wholly vegetarian dietary is the best throughout. The writer cannot indorse this as an absolute or general rule. He has seen cases which certainly have done well on such a diet, but on the whole a mixed diet of flesh, vegetables, and fruit has been the most satisfactory. Some have their strength completely restored without meat, but in America and England, under present social conditions and habits, the mixed diet has proved most applicable as well as most serviceable. In warmer climates the less meat the better. There is much more therapeutic potentiality in diet than is frequently thought, and the best kind of food needed for each patient deserves the special attention of the medical attendant. In an ideal state, however, the writer would, if he could, secure proper cooking and command a judicious selection of articles of vegetarian diet, preferring to rely on a vegetarian diet with little or no flesh meat. A word of caution as to oatmeal. In the South of England and in warm countries this excellent food-stuff is apt in many persons to cause heartburn, waterbrash, and other symptoms of indigestion, with a peculiar uncomfortable irritation of the blood and skin blotches, when its use should be interdicted. Wheatmeal may be substituted. Though the writer has not seen juicy fruits avert drunkenness, he has seen them aid powerfully in the cure of inebriety. The natural fruit juices tend to allay if not to quench the great thirst, which some inebriates experience when they are in the throes of the dyspeptic miseries following a bout of intoxication, besides having a depurative influence on the blood. Nutrient enemata may be required, though the writer has not had occasion to resort to them. If delirium tremens be feared frequent quantities of light nourishing food with a simple mixture of calumba, aromatic spirit of ammonia, in 10 to 15 minim doses, and bicarbonate of soda have often helped to avert an attack. In the stage of gastric irritability with emesis, large draughts of water should not be given as they are apt to produce vomiting, but sips of cold and still better hot water are grateful. Some derive the most benefit from sucking ice occasionally. During this stage tonics should be sparingly employed; but they are a sheet-anchor afterward. Nux vomica and strychnine are the most generally useful. They clearly exert a great influence in bracing up the nervous tone, and if prescribed with due caution are as safe as they are potent. Combined with either an acid or an alkali they are often more useful than when given alone. Valerianate of zinc is a good adjunct, and may be with advantage combined with pepsin and sulphate of iron. The writer has occasionally found calumba, quinine, salicylate of quinine, and sulphate of beberine, among other remedies, of considerable value. For a host of various medicines and therapeutic combinations extraordinary curative and prophylactic properties have been put forth. The composition of some of these has not been disclosed, though it is probable that a fairly accurate approximation to the truth has been arrived at. The composition of others, employed by medical practitioners practising in accordance with the recognized code of ethics of the profession, has been therefore freely proclaimed. But whether disclosed or undisclosed, the claims urged on behalf of any drug or drug-combination that it is an absolute or nearly absolute cure, in the writer's opinion may at once be dismissed. There is and can be no specific for the "cure" of alcoholism, and any medicinal or other preparations for which such a claim is alleged in all probability have been long found useful, not as specifics, but as serviceable in improving the tone of the brain and nervous system. It is probable that strychnine or nux vomica will generally occupy the chief place in most modes of treatment open or mysterious. The greatest danger in its secret employment lies in the fact that the doses to be taken by the chronic alcoholic are not known; strychnine, though of the highest value in moderate doses, being in too large doses perilous to life. Among many other drugs which have been employed are chiretta, gentian, iron, manganese, chloride of gold and sodium, hydrastis, phosphates and phosphites, muriate of ammonia, atropine, cocaine, cannabis, capsicum, and antimony. The mode of administration merits consideration. The method by hypodermic injection has been highly lauded by Portougaloff and other orthodox practitioners, and is used in some of the secret "cures," but in the writer's opinion is not a desirable plan in the ordinary run of cases. Probably the puncture for a short time makes a greater impression on the mind of the patient, but there are risks involved, in this seemingly simple operation, which he thinks should not be run, especially as the therapeutic virtues of drugs can be as efficiently imparted by the mouth. Besides this, subcutaneous self-injection has a remarkable fascination for a certain type of mankind, and the writer has known serious mischief arise in this way. The writer prefers official pharmacopœial preparations in liquid and pill, but he also employs tabloids, perles, and bi-palatinoids. He has never seen any harm follow the use of alcoholic tinctures in judicious doses, so he has not used glycerin tinctures, his objection to the last-named being that this substance does not extract all the medicinal virtues of some drugs and cannot be employed with others. To any physician who fears any detriment to a victim of alcoholism from alcoholic tinctures, the glycerin form of a large variety of drugs will prove serviceable. In treatment, hypnotics are at times required. As a whole, in chronic alcoholism, the writer used to find the bromides with henbane and chloral hydrate or croton-chloral hydrate in small doses generally answer well, but of recent years he has experienced better results with phenacetin and trional. In violent cases where subcutaneous injection is imperative, he looks to morphine, hydrobromate of hyoscine, gr. $\frac{1}{200}$ (injectio hyoscinæ hypodermica, B. P., 1 minim) or hyoscyamine sulphate gr. $\frac{1}{120}$. If, however, he can secure wet packing he prefers that to any other procedure. In most cases, though not in all, this process is effectual, and has the immense advantage of being free from risk. Unfortunately it requires to be intelligently applied and necessitates attendants in acutely violent cases, hence is not always practicable in a private family. Special attention must be paid to other diseases complicating chronic alcohol poisoning. Syphilis has been present in a substantial proportion of the writer's cases, and is frequently met with in different countries. A mercurial and alterative course of medicine is called for here in addition to the treatment directed against the alcoholism. Neurasthenia calls for such remedies as arsenic and iron; malarial troubles (rare in England but frequent in America), for quinine, beberine, or arsenic. An enteric factor necessitates special diet, cardiac symptoms may need digitalis, strophanthus, or sparteine. A word of caution may be interposed on the desirability of watching the effect of medicines, especially such as are powerful. Strychnine and atropine for example, unless given (as the writer prefers to give them where they seem to be called for) in moderate doses, may end in disaster. Heroic administration for a sensational *coup*, in the writer's opinion is a perilous procedure. It is true that what is popularly called the "drink-crave" may for the time in some persons be broken up; but in not a few cases at the risk of grave nervous and cardiac injury which may prove irreparable. In the few cases in which for special purposes of urgency the writer has resorted to this, he has always felt great anxiety and has insisted on explaining the risk which the patient would run. Yet he has never employed the doses which have been said to have been given, almost as a mere matter of routine, by some practitioners. His observation has been that the relief has generally been temporary, often remarkably evanescent. Even with such comparatively safe drugs as potassium bromide, he has seen lengthened and even permanent damage done, particularly to the brain and nervous system, by the heroic doses given designedly by some physicians with the best intentions. He refers to such heroic doses as 60 to 100 grains and upward. In this drug his observation has been mainly of Englishmen and Englishwomen, who have been so treated for disease other than, or complicating, alcoholism; having had to deal with distressing cases of shattered nerves and broken-down constitutions from this cause. He therefore desires to impress on the medical mind the great importance of noting the effect of powerful drugs, especially on the nervous system and cardiac apparatus of inebriates. There is the greater reason for the most guarded and moderate employment of powerful remedies in this disease, inasmuch as the heart muscle itself in chronic alcoholism is pretty certain to be flabby, weak, and loaded with fat, muscular fatty degeneration being a constant effect of persistent free alcohol consumption. This is apart from the cardiac debility resulting from the frequent excitation and disturbance of the heart, almost synchronous with and arising from the nervous riot caused by the ingestion of alcohol. The organ, too, under free alcohol soon begins to expand, leading on to a feeble dilated heart. This relaxed cardiac dilatation, with the loss of elasticity and atheromatous increase of rigidity in the arterial system, and the venous distention contribute to form an abnormal condition of the heart and circulatory apparatus both of which exhibit a delicate susceptibility to the action of strong remedies such as strychnine and atropine. If this caution is imperative on the orthodox physician who unreservedly prescribes in writing his medicines in definite doses for each patient, after studying the individual symptoms and idiosyncrasy, how much more imperatively is it called for by persons who, bound by no allegiance to the recognized code of ethics of the medical profession, produce and issue preparations or mixtures alleged to be specific for the cure of alcoholism! To be of any real value preparations for this purpose must contain drugs of some potency, yet they are sold indiscriminately to all who purchase them, so that they are taken without any consideration of the individual's cardiac health, the feeble in heart drinking the same dose as the strong of heart. In Great Britain this is unavoidable, for the exact contents of commercial or patent remedies are not printed on the exterior of the package or bottle, a practice which, with a more conspicuous statement of poisonous substances where these form part of the ingredients, would tend to lessen the risk of disaster. The popular and loud demand for something which would at a blow destroy the overpowering drink craze or impulse which dominates so large a proportion of chronic alcoholics has produced a host of alleged "antidotes" many hundred strong, which, as in cancer and other ailments, are expected to be effectual in all or nearly all cases. The many victims of alcoholism who have tried numberless "specifics" in turn, who have taken the teetotal pledge over and over again, or who have even publicly testified to religious influences or drug preparations having destroyed their drink-crave so utterly that they could never drink an intoxicant again, and yet who have returned to their former alcoholic indulgence, negative the existence of such an antidote; while the hundreds of thousands of reformed drunkards, who by moral, religious, or selfish motives alone have overcome and stood firm in abstinence, attest the fact that confirmed alcoholists can be and have been cured by a resolute effort of will. Simple remedies are often of service in pacifying for the time the morbid intoxication impulse or craze. An orange, a cup of coffee or cocoa, a glass of hot milk, for example, will answer the purpose with many; while others are better served by 15 to 30 drops of tincture avena sativa, or 5 to 10 minims of tincture of chloride of iron in infusion of calumba. At times the writer has found an emetic of ipecacuanha act well. An apomorphine hypodermic injection of from 2 to 6 minims of the official injectio apomorphinæ hypodermica (2 grs. in 100 minims of camphor water) would answer the same purpose, and could be administered without the patient knowing the effect of the drugs the first time; but the writer prefers that the patient should take medicine intelligently when conscious, as on the exercise of the intelligent will-power the hope of his cure mainly if not entirely depends. Hypnotic suggestion has been highly spoken of by some in quieting and preventing the drink impulse, crave, or craze, but the writer has seen so many alcoholics with whom this process has failed, that, apart from the many objections which have been fairly urged against it, he cannot recommend hypnotism. Making every allowance for the influence of concurrent factors, he has seen morbid symptoms of increased mental instability and will-palsy which he could not attribute to any other cause than artificially produced hypnosis. Hypnotism, which is identical with mesmerism, being simply a new name for the remarkable phenomena exploited heretically (medically speaking) by Mesmer, and afterward in orthodox medicine by Braid and Gregory, claims to do no more with alcoholists, though certainly it now, in the ranks of the profession here and there, asserts itself more loudly, than did the mesmeric treatment of Gregory; but though this amiable and earnest enthusiast fully believed that he had cured the disease, this belief having been shared by many of his fellow-citizens in Edinburgh, the apparent wondrous "cures" passed away and the transient popularity of mesmerism faded there without, at least for many years, leaving a trace behind. Similar and even more apparently marvellous cures on a wider scale, said to have been wrought by either some stated or mysterious chemical or medical preparations or processes, have in turn "held the field" and in turn followed their predecessors into history. It has also been claimed for hypnosis that it has been of value in strengthening the alcohol-paralyzed will and thus effecting a "cure."

The writer has never seen this, though, as under every conceivable condition and circumstance a certain proportion of curable alcoholists will exert the will-power remaining to them and cure themselves, he has always been prepared for a certain ratio of permanent recoveries after hypnosis, or any open or secret "specific," or without the taking of any drug or submission to any process, or treatment of any kind. He has known an alcoholized pair, a man and his wife, very bad cases of several years' standing, influenced by no moral or religious motives, but purely from a determination to "better themselves," suddenly stop drinking and thereafter lead a life of abstaining sobriety. He has studied a large amount of evidence given by acknowledged medical authorities on hypnotism, and cannot from their reports, when compared with the actual state of things in the surrounding population, extract any proof of the true abiding usefulness of hypnotism in the treatment of alcoholism. The hypnotic mental unsettlement and impairment of will, as well as the influence which the hypnotizer can if he choose cultivate and exert over many of the subjects, constitute in the writer's opinion a fatal barrier to the ordinary employment of hypnosis with inebriates; yet it is only fair to add that success has been claimed by some. Hypnotic suggestion in this malady has been applied for two purposes, one to suggest to limited drinking as by Liébeault and Bernheim (which in the writer's opinion is altogether a mistaken purpose), the other, as by Forel, to suggest to entire abstention, which in the writer's opinion is the essential object to be aimed at. The cases recorded as having been treated hypnotically are far too few to justify a recognition of this process being so generally effective as to warrant it being classed as curative of the disease, leaving out of question the serious drawbacks to its employment even if proved effectual. The writer ought perhaps to add that he was for a time, about a quarter of a century ago, an enthusiastic believer in the value of hypnotism therapeutically, but a brief experience soon showed the uncertainty, unreliability, and resulting brain and nervous disturbance so plainly, that he had no alternative but to accept the disillusion. It ought ever to be remembered that many inebriates, especially periodic inebriates, are practically, during all their alcoholized career, running in psychological cycles. There is first a gradual (sometimes sudden) term of inebriate excitation and access, then the alcoholic explosion, followed by a term of inebriate decline which often is prolonged in a longer or shorter term of strict abstention. During this abstaining interval the repentant subject of this inebriate storm may be as earnest and ardent a temperance advocate as any abstainer in the land, by and by so exhausting his energies by his tremendous overwork and overstrain as to induce resort to an alcoholic pick-me-up, when the whole narcomaniacal round of periodicity is gone over again. In this inebriate cycle the abstaining term is as really a part of the cycle as is the alcoholic explosion in a bout of prolonged intoxication. Inebriates, who seek the hypnotic or any mysterious remedy or process which has been claimed to have worked no end of "cures," are, too, in the stage of recess and depression, in the very psychological state most ready to be impressed by oratorical appeals to the imagination and glowing promises of the hope of recovery. In judging of the legitimate effect of any decided improvement it is imperative to bear in mind the typical temperate intervals of the periodic inebriate's cycles of inebriism. It may not be amiss to refer to the extraordinary mental temperance waves or revivals which have, every now and again, passed over a whole state or country and have apparently swept away in their ardor for abstinence all the alcoholists in the community. The millions, including nearly all the drunkards, to whom Father Mathew administered the teetotal pledge in Ireland, about fifty years ago, besides hundreds of thousands in England, Scotland, and America, bade fair to make the Irish a perfectly sober nation. The number of distilleries was rapidly diminished by one-half. But a small proportion of the pledged remained steadfast, and the people have long since returned to their former indulgence. So with the Washingtonian and other movements in the United States. These neurotic temperance waves have all flowed and ebbed, leaving behind them on the sands of the nations, as they receded, a fair salvage, though but a small proportion of the original number of reformed drunkards carried away at first. We need not be surprised therefore at the success which has been claimed for hypnotism, which appears in neurotic waves, passes over certain sections of the peoples after intervals of years, impressing subjects of nervous maladies like narcomania, with a lively hope of relief. There is, however, a form of mental suggestion which may safely and usefully be employed. The really successful physician is one who, by his influence over a patient, inspires the latter with hope and impresses on him the value of the counsel, the patient all the time being in a state of expectant mental concentration on the physician's words. This is quite different from hypnosis, and is simply intercourse between two persons both in the full possession of active mental capacity. No one can doubt the importance and the propriety of non-hypnotic suggestion when intelligently applied. One remark applies to all rapid "cures" or processes observed or made known without reserve in all their particulars. A diseased condition of brain, such as narcomania is, cannot be remedied in a few days or weeks. Symptoms may be and are alleviated by ordinary considerate yet firm care or by a friend taking the law in his or her own hands, as the writer has known a wife do by locking her drunken husband in a room for days, sometimes after administering a thrashing. So can a similar improvement be wrought by kindness and care for a week or two, without medical treatment. And a certain proportion of inebriates thus dealt with non-medically will continue the improvement and remain abstainers. In from seven to sixteen days nearly all thus helped are ready to testify that they are "cured," but experience shows a preponderating majority of relapses. The mere fact of a rapid "cure" being claimed ought to call forth strict caution and deliberation, especially in cases of long standing. Weeks and even months are required for the elimination of the poison and the correction of the actual mischief done to organ and tissue, during and after which the structure must be built up anew of sound healthy tissue, a labor of time. If all this can be accomplished when the patient is following his usual calling, so much the better. The having a daily duty to perform and the performance of it keep the mind concentrated on the task and less liable to temptation to drink. But, unfortunately, the chronic alcoholist is rarely brought for medical advice till the indulgence has become so crystallized in the daily life, that the will-paralyzed inebriate needs external aid to protect him from temptation. He has, as a rule, not invoked medical advice till after the family clergyman and lawyer, and probably most of the friends, have been appealed to. Thus he has often had a chronicity of from three to four or five years up to fifteen and twenty years, before the physician has been called in. The duration of the indulgence has often so confirmed the disease that the will is practically powerless, and the patient needs all the aid possible to protect him against himself. In such cases, as with the insane, a change of environment with its new associations sometimes of itself has a beneficial influence, but the main immediate necessity is the absence of intoxicants and the inability to procure them. When the alcoholist has to be sent from home, two points of first importance confront the medical adviser when his counsel is sought, though very often the inebriate and his friends prefer to act on the advice of their lawyer, or clergyman, or some other amateur, neither of whom understands the disease and the treatment required nor is therefore competent to give intelligent counsel. One point is the character of the home to which he is to be advised to go. This is the consideration of greatest moment, but it is very generally overlooked. True, even in a lax institution where neither discipline nor the intelligent and discriminative medical treatment of each case is exercised, or still more in a place where drinking is connived at for mere pecuniary gain, some inebriates will be cured by their own reassertion and exercise of will-power; but the responsible physician will feel it an imperative obligation to recommend, if this be within his power, only an establishment which is intelligently conducted on lines in accordance with the recognized ethical code of the medical profession. There may not be a doctor at its head, but, whoever the proprietor or whatever the proprietary, the treatment should always be directed by competent and straightforward medical practitioners. Whether under medical or lay auspices, in no circumstances is it desirable for an inebriate to be placed in a home where intoxicants are used by any one as a beverage. Such a retreat or hospital, public or private, should be a prohibitory territory where alcoholic liquors, if employed at all, should be medically prescribed with as great, and even greater, caution than any other narcotic anæsthetic. If the medical staff and other officers are abstainers, so much the better. The writer cannot understand how any enlightened medical practitioner, who has the well-being of his or her inebriate guests at heart, can remain a non-abstainer. The quiet example of the resident physician and officers, or of the host and other members of a private medical household taking such a patient, has often a deeper influence on the afflicted inmate in confirming his good resolutions and strengthening his will than any amount of prescriptions and physic (though all are needed); while the innate cunning of the alcoholized individual unconsciously—it may be, against his own intention—adapts the medical adviser's practice of "moderate" drinking to the heightening and confirming of the morbid desire for or impulse to intoxication. These remarks apply with equal force to attendants or companions of inebriates, and to all persons under whose charge in any way these cases are placed. In addition to the genuine character of the home or person to whom an inebriate should be entrusted, the suitableness of the locality should be carefully weighed. For instance, it would be unwise to send a consumptive alcoholist to live over a damp clayey soil, or a rheumatic inebriate from a dry airy gravelly region to sojourn in a close valley reeking with moisture, where there are frequent and dense fogs and wet, muddy roads. The climatological idiosyncrasy of the patient has also to be considered. Some alcoholists, especially those of the irregular periodic type, while sober when at home, say in a locality in the interior, in a few hours after going to the sea-coast experience great nervous perturbation, which is controlled (though only for a few hours, after which the neurotic storm begins to brew again) by intoxication. These subjects feel calmed by the first glass of spirits, but with spasmodic rapidity the temporary calm yields to a renewed and intensified nerve-paroxysm, and a drunken bout is the issue. If they awake from that, and remain by the sea, the nerve-excitation recommences, with a repetition of the alcoholic nerve-whirlwind. Yet these very sufferers, just as if they were asthmatics whose distress is aggravated and heightened by leaving the coast and going to a rural district in the haying time in summer, if they retire inland, are at once freed from their nerve unrest, subside into their usual routine, and lose the impulse to inebriate excess. These, with other forms of spasmodic neurotic overpowering disturbance, such as with some the transfer from a damp to a dry soil, are exceptional cases, but the possible latent presence of such conditions has to be reckoned with. A very common obstacle to treatment is urged now. It runs to this effect: "My son has inherited this disease and nothing can cure him." The patient himself often says: "I have inherited it. It is no use my trying. You cannot help me." This is one of the fruits of so much having of late years been written and said of disease-inheritance. But it is a popular misconception from the confusion of disease with tendencies or susceptibilities to disease. It does not follow that because a child has intemperate or gouty parents, he must necessarily suffer from drunkenness or gout. Granting a gouty or inebriate inheritance, he has a preponderating chance of never being afflicted with either disease if he is brought up and continues under conditions unfavorable to the development of the symptoms of either malady. If he be trained as an abstainer and continue an abstainer his chances of becoming a drunkard or a gouty individual are very slight. Further, by appropriate, sound, wholesome fare and regular moderate hygienic exercises, his system may be so strengthened and fortified as to throw off these transmitted tendencies. Even though the patient may have a direct alcoholic or other inebriate ancestry of two or more generations back, he need not despair. Time and again, the writer has seen cases with the gravest family history as to inebriety and with other neurotic family trees of serious import; yet these handicapped persons, though they have been confirmed in their alcoholic intemperance for long terms of years, have made a good recovery. It ought to be an imperative obligation binding on the conscientious and enlightened physician to assure his inebriate patients that their cases are not desperate; that if they have inherited some weaknesses they have also inherited other strengths, and that if they follow wise counsel they have good reason to expect a permanent cure. In no one part of the medical treatment of the inebriate is the skilled physician's advice more urgently called for than in the encouraging of the despairing patient with a strong hope of cure, which indeed has been achieved in one-third of all the cases which have been under recognized and judicious medical care.

The alcohol having been thoroughly eliminated and its pathological sequelæ having as far as possible been removed (in a proportion of cases these can be reduced only to a minimum, as in cirrhosed liver or alcoholic fatty heart), the exciting and predisposing conditions which have been found to have contributed to the disease must be grappled with. At as early a stage as possible in the treatment this should be attended to, especially as regards the excitant influences. Some of these contributory factors will have ceased to be, in new surroundings, as in the case of inebriate excitation from association. A wine merchant or a publican who is continually subject to temptation, or a commercial man whose business intercourse has been daily cemented with alcoholic intoxicants, will at once be freed from these excitant circumstances in a home or retreat where no such liquors are allowed. But there are other exciting causes which require looking after. For example, idleness should be corrected by setting the inebriate patient to do a certain amount of muscular or mental work every day, the physician having first determined the kind of occupation best adapted for the particular patient. Again, if rheumatism, or neurasthenia following such a malady as enteric fever, or specific disease, or any other unhealthy physical condition has been a causative factor, appropriate remedies must be employed to improve these diseased conditions. In women some uterine displacement or affection, some ovarian complication or some menstrual disturbance may have been a chief feature of the causation. In such cases the misplaced organ should be replaced, and the exciting morbid disturbance rectified. In this connection, massage, electrical stimulation, Turkish and other baths are excellent adjuvants to therapeutic medication. It has been claimed for the Turkish bath that it is a "cure" in itself. There could not be a more mistaken belief, and the extravagance of the claim has somewhat lessened the reputation of the bath. Hydropathy has likewise been held up as a "cure" on equally baseless grounds. Both of these bathing processes are of inestimable service when moderately and judiciously applied, in combination with other remedies. The perspirative elimination of unduly retained waste-products, the flushing of the emunctories, and the soothing influence on the nervous system, of the hot-air bath and the wet pack are grateful and useful to many inebriates. So are the shower bath and the ordinary cleansing tepid or cold bath. But these must be supplemented by treatment directed to the reparation of sound body, brain, and nerve tissue, to the restoration of inhibitory power, and to the re-establishment of the will. In no single process, appliance, or drug is there to be found a specific for alcoholism, though if one were determined to proclaim a "cure" it would be very much better and safer to employ such open modes of treatment as bathing processes than undisclosed preparations which might or might not be composed of potent and dangerous drugs in doses of which the strength was not known. The writer is most anxious to avoid the imputation of attempting to throw discredit on Turkish baths and hydro-therapeutics, which he prescribes, and from which he has derived not a little pleasure and comfort when attacked by various ailments. His intention is to locate their true place in the therapeutics of inebriety. One of the conspicuous symptoms of alcoholism (more pronounced in the alcoholic than in any other form of inebriety) is a failing regard to personal cleanliness, which is most marked in women; and, as in melancholia, one of the first hopefully prognostic signs is a reawakened interest in personal ablution. Hence the great importance of insisting on baths being taken.

The prescription of wholesome good food, partaken of at regular hours and consumed slowly, is important. Though it may seem trivial, the writer has seen occasion to direct attention to the state of the teeth. Thorough mastication is essential to proper digestion. What has already been stated about food-stuffs is as applicable to the continued reconstruction of the system and the renovation of permanent brain and nerve texture. A properly selected dietary should be directed for the needs of each patient. A man doing steady muscular exercise requires more solid food than a woman, merely sewing and playing, while the tendencies to certain other maladies call for the avoidance of certain articles, such as sugar in diabetic cases. Drugs do not play so leading a part in the later as they do in the earlier stages of treatment. Yet the public and the patients generally labor under the delusion that there is or can be no medical treatment without drugs. The enlightened physician knows that medicines, in the case of the inebriate as of the insane, form but a part of his therapeutic resources. It often requires more skill to withhold than to order physic. Yet where this demand is manifest (a demand typified in the person of miners in certain localities in Britain, who unless "worked" by the strength of the physic, as in former times "blooded" by the lancet, think the doctor is "no good"), it is unwise to refuse the psychological support of a harmless dose. Such patients are so disappointed at not having to take medicine, that the refusal of it depresses their spirits and damps their hopes, thereby affecting the prognosis and retarding if not diminishing the prospect of their cure. In such cases, accordingly, the writer has felt it his duty to prescribe some useful and safe tonic in restricted doses, and has found such combinations as the following of decided service: In pill, ferrous sulphate and compound asafœtida pill, of each gr. $\frac{1}{2}$, twice or three times a day, or 5 minims of dilute nitro-muriatic acid with infusion of gentian, or ammoniated tincture of valerian with infusion of calumba. There are, however, many cases which call for and derive great benefit from long-continued medication, in addition to air, exercise, occupation, and general hygienic measures. For such, as a general rule, by far the most useful remedy is some preparation of strychnine. One minim of liquor strychninæ or gr. $\frac{1}{50}$ of strychnine nitrate, or strychnine hydrobromate, or $\frac{1}{100}$ of a grain of strychnine arseniate may be given once daily. Another good preparation is citrate of iron and strychnine, the triple citrate of iron, quinine, and strychnine, in 2-grain doses, twice or three times daily, sometimes answering well. Nux vomica has, in the writer's experience, been of value, given in 5-minim doses in an acid or alkaline mixture twice a day, or in a pill in the dose of one-sixth of a grain of the extract. With dilute nitric acid and taraxacum, nux vomica is specially valuable in a large proportion of cases such as those with hepatic disturbance, and with potassium bicarbonate (in both sets of cases along with a vegetable bitter infusion), and perhaps potassium iodide, in cases with a rheumatic diathesis. With strychnine and nux vomica, other combinations are indicated for other disease tendencies and complications. A strong note of caution should be here interposed. Even such moderate doses as have been just stated should never be given for a long time, say more than six weeks or so, without a break. Some constitutions seem to be peculiarly susceptible to the influence of such potent and so generally useful drugs as strychnine, and it is judicious therefore to watch the effect of the remedies prescribed, to note their characteristic action on the individual patient (as when strychnine is pushed too far, depressing the nervous system and weakening the heart's action) apart from their influence on the disease itself. Even when such a powerful drug as strychnine has been given in such small doses as gr. $\frac{1}{60}$ once or twice daily, the writer has found it sometimes advantageous to reduce the dose or substitute a less risky nerve tonic for a time. With this and other drugs some of which if pressed too much and too long might induce nervous disorders, possibly including insanity, this caution is necessary; but even with drugs which in excess would produce probably only physical discomfort, and may therefore be deemed to be innocuous, a change is occasionally desirable, many medicines losing their beneficial influence after a time. Among a multitude of medicinal remedies contributing to the rebuilding of healthy brain and nerve structure many of the preparations of bark and quinine hold a high place, while good results have also been claimed for calumba, gentian, chiretta, aurantium, the hypophosphites and hypophosphates, phosphoric preparations such as dilute phosphoric acid, iron and manganese combinations of which liquor mangano-ferri peptonatis is one of the newest and best. Gold is specially favored by some physicians, and is a heightener of nerve tone, in the writer's opinion inferior to strychnine, arsenic, and iron, or even ordinary vegetable bitter tonics. Massage is useful in a small proportion of cases, where the absence of muscular exercise in walking (especially in the open air) cannot be enjoyed, but is a poor substitute. Its chief value is seen in neurasthenic females, till they have recovered so far as to be able to have pedestrian or equestrian exercise. Electricity and faradaism are in a similar category, of service in a few exceptional cases and for a limited time. For all classes of patients and for all the varying forms of the disease, cleanliness with muscular and mental exercise, after a transient period of rest, come next to abstinence. After the system is freed from alcohol and its sequelæ, the recovered muscular energy is so great that, especially with males, it is best directed into a safe channel by hours of walking exercise, or by hard manual work in carpentering or at the lathe. But after a shorter or longer period this intense accumulated muscle-energy is quieted down to a steady supply. So with brain-force. In the advanced stage of revived regular normal body and brain capacity systematic exercise of body and brain is essential to the permanent soundness of both. Men and women present difficulties peculiar, so to speak, to their sexual difference. The former can have walking and riding, manual toil, and manly exercises; the latter, walking with lighter occupations such as sewing, wood-carving, and the like. Both sexes can read, write, draw, paint, photograph, play on musical instruments, and so on. Among the mechanical classes and the poor, inebriate men can dig, garden, do house decorating, and other remunerative work, while inebriate women can wash, iron, knit, sew, and do housework, and a hundred other light duties. Alike for rich and poor, it is of the first importance that they do not lead an idle life, the old proverb being true, that "the devil tempts a busy man, but the idle man tempts the devil." The methodical occupying of the mind with the performance of daily duties is the only effectual means of withdrawing the inebriate from his morbid brooding over his misdeeds and miseries, and thus powerfully aiding in making him once more "a whole man" in fair possession of self-restraint and inhibition. Tobacco, especially when smoked, has been stated to have been useful in the treatment of alcoholism, most Homes allowing males a pretty free license in smoking. It cannot be denied that there are alcoholic inebriates who can for the moment stave off a paroxysm of inebriate impulse or crave, some, if they can smoke for an hour or two, actually blunting the drink impulse or crave till it has subsided; but this transient benefit is secured only in a very small proportion of alcoholists, and is dearly purchased. While tobacco, even to those who are thus soothed by it, procures temporary relief, it is apt on most patients to inflict considerable substantial injury. There are many exceptions, male and female, who appear to thrive on tobacco; but as a rule, particularly in inebriates of neurotic origin, depression is an after-consequence, and the heart's action is enfeebled. In some cases, the cardiac and nervous depression induces a tendency to faintness which calls for a recumbent posture and prompt stimulation by hot coffee or hot water with a little ginger, or the application of heat to the feet, most intense if the patient has been smoking on an empty stomach. To all such cases tobacco should be strictly forbidden. Others have their digestion disordered, their throat congested, or their vision impaired. To these also smoking should be interdicted. The routine practice of permitting smoking is more honored in the breach than the observance. If allowed at all, at best a doubtful practice requiring close observation of the effect produced on each patient, smoking should be regulated by the physician according to the condition of the smoker. Acknowledging to the full an occasional temporary assuaging of the inebriate paroxysm in a few cases, the writer is of opinion that even for this purpose a more innocent remedy may be found, and that the use of tobacco has no legitimate place in the curative treatment of alcoholism, while tending on the whole to retard convalescence. The only defence of a permission to use tobacco in such cases seems to him to lie in the lessening of the discomfort to alcoholic inebriates who have been tobacco users.

It will have been observed that the writer has indicated only the lines on which sound treatment can be based, with suggestions as to the proper application of medicines and remedies. No one pharmaceutical combination or curative process or therapeutic adjuvant is suitable for all cases. To be intelligently treated each case must be studied by itself, apart from, as well as in comparison with, other cases. The mixed forms of the disease of narcomania present an endless variety of symptomatic combinations, while the idiosyncrasies of many an individual constitution are so mingled with the alcoholic phenomena as to constitute a puzzle, the unravelling of which demands a patient and accurate study. This unravelment once accomplished, by a correct diagnosis having been arrived at, the scientific physician will be in a position to apply the resources of the "art of healing" best adapted to the case under observation. In all cases the treatment will not be complete unless the highest part of the patient's nature receive due attention throughout. The reason ought to be enlisted in the treatment, which is best attempted by sound teaching concerning the fallacy of the prevalent belief in all classes of society, in the virtues of alcohol as a beverage, and concerning that potent drug's benumbing influence on the senses, its undermining action in structurally degrading organ and tissue, its irritant inflammation of the texture of vital organs, its riotous disturbance of function, its diminution of muscular power, and in many cases its demoniac possession of the entire man. He should be especially grounded in the truth as to the non-necessity and valuelessness of alcoholic intoxicants as an article of ordinary diet. No pains should be spared to impress on him the need for his persistent abstinence from all intoxicants as long as he lives; and all the influences of culture, music, and the fine arts, of high-toned morality and pure undefiled religion, should be enlisted to strengthen self-respect and fortify volition and inhibition. Alcoholism should be treated at an early stage of the disease. In chronic alcoholic, as in chronic plumbic, poisoning, the cumulative effects of the poison necessarily increase with the duration of the poisonous process. As Lallemand, Perrin, and Duroy have shown by their experiments, alcohol has a tendency to accumulate more in the liver than in any other organ, and accordingly the liver is the organ which *par excellence* has disclosed on post-mortem examination distinct alcoholic lesions. The brain and nervous system, too, are more injuriously affected the longer this brain and nerve poison is employed in poisoning nerve tissue. Besides all this, repeated acts and states have a tendency to leave behind them a memory which develops into automatic performances done practically without conscious intention. In addition there is the anæsthetic narcotizing property, inherent in alcohol and its inebriate congeners, of creating an appetite for a fresh supply. Therefore when alcoholism assumes a chronic character it is the more difficult of cure the longer has been the accumulation of the morbid disturbance and tissue changes. It is a much more arduous undertaking *cæteris paribus* to treat an alcoholist of ten to twenty years' standing than of from one to three years' standing. Hence treatment should be applied at as early a stage as possible, an axiom which most alcoholists and their relations fail to act upon.

Ginger, Eau-de-Cologne, and Lavender Inebriety.

Habitual indulgers in excessive quantities of ginger alcoholic preparations, eau-de-Cologne, and lavender are all practically alcoholists, and the pathological effects of each of these drugs are those of alcohol poisoning, slightly modified by the properties of the particular preparation, in the same way though to a lesser degree, as the effects of alcoholism are modified by the particular beverage; for example, beer has not quite the same action as whiskey, brandy, or absinthe, though all these four liquors are productive of alcoholism. Alcoholic ginger preparations are the most injurious of these three sub-varieties of alcoholism. The form in which this drug is ordinarily consumed is essence of ginger (tinctura zingiberis fortior, B. P.), a spirituous preparation of nearly twice the alcoholic potency of brandy, or whiskey, containing 84 per cent. of absolute alcohol. The writer has seen several cases. One male case was a barrister (with immediate paternal alcoholic heredity) who had been a periodic alcoholist for twelve years. He latterly suffered so acutely from gastralgia and mental depression as to all but drive him to suicide; but, finding temporary relief from essence of ginger he insensibly became addicted to "ginger drinking." He began with ten drops occasionally and in three years advanced to one pint per diem. While the prior alcoholic inebriety was periodical and social, the latter ginger-with-alcohol indulgence was constant and solitary. Even in courts of law he indulged furtively. In the alcoholic period he suffered less pain and lost his suicidal melancholia, though his narcomaniacal slavery became more confirmed. The pathological chain of events in this case was neurasthenia, brain and nerve exhaustion from mental overwork, combined with insufficient air and bodily exercise. Atony of the gastric nerves was the result, with alcoholic partial destruction of the mucous membrane of the stomach, causing neuralgia in that region. As a carminative the ginger comforted that organ, while the increased alcoholic strength enhanced the alcoholic anæsthesia. Female cases have exhibited a similar history in the main features, though in some cases there had been no gastric trouble beyond dyspeptic discomfort, and in a few others no special stomach derangement. The chief symptom in females was a feeling of a constant gnawing in the pit of the stomach, generally from frequent "nips" of ardent spirits with excessive drinking of strong tea. Other ginger alcoholic preparations are prepared with cheap spirits, sometimes involving the consumption of the heavier alcohols with their added poisonous effects. Methylic alcohol (wood spirit) is often employed on economical grounds, and is quicker in producing intoxication than ethylic alcohol. Crothers has noted in the United States that females go on from ginger to purely alcoholic indulgence, ginger addiction having been the forerunner of alcohol addiction. In Britain the writer has observed that both sexes begin with alcoholic drinks and end with ginger alcoholic compounds. The quantity consumed by gingerists is sometimes so large that one American druggist sold to a dozen regular customers from one quart to two gallons weekly. These alcoholic ginger liquids must not be put in the same category with the increasing supply of non-intoxicant beverages slightly flavored with ginger, which are to many persons an agreeable warming refreshing drink, especially in winter, not only enabling one to withstand the alcohol-crave but also helpful in the cure of a considerable proportion of inebriates. Ginger inebriety is more general in the United States than in Britain and elsewhere, but in the latter country it is also on the increase.

Capsicum addiction is a more advanced form of alcoholic indulgence. The alcoholic tincture is taken constantly and in large quantities, as by an inebriate officer in the British army, aged forty-six, who after having drunk to excess for many years became so inveterate a capsicum taker that he added tincture of capsicum to every draught of intoxicating liquor, and to liquid dietetic dishes such as soup. The gastric depravity induced by the capsicum in combination with the alcohol so changed his disposition that the erewhile cheery, active, lively, amiable companion became prematurely aged, a wretched melancholy, snappish misanthrope. Thus a drug which is exceedingly useful in the treatment at certain stages of alcoholic inebriety in men exposed to the air in wet weather and irregular in eating, becomes to some inebriates the means whereby they indulge in intoxication.

Eau-de-Cologne is another strongly alcoholic preparation which is a convenient and easily taken intoxicant. As it is *en règle* on a lady's toilet table, its presence there does not excite suspicion, and unfortunately the readiness with which it can be taken on a piece of sugar adds to its seductiveness. It is at first usually taken in this way, but many women are so addicted to its internal use that this mode is not satisfying enough, so they drink it by the spoonful with and sometimes without water. North America and Britain are the countries where eau-de-cologne is most extensively employed as an intoxicant. In the latter country, especially in large cities and among the wealthier families, the spread of this variety of spirituous indulgence has attracted considerable attention. In the rapidly increasing demands of fashionable social duties with the tremendous nervous overstrain and consequent exhaustion, a resort to eau-de-Cologne secures a temporary if fictitious stimulus, imparting to the wearied hostess and the jaded guests an animation for a time, which is welcomed by both. The result is as disastrous to the physical health as it is so often the overture to a career of inebriety. Young girls are especially liable to be undermined in health and sobriety in this way, while elder matrons too have thereby become alcoholic wrecks. This addiction is also penetrating into the ranks of domestic women of a physically or mentally feeble type, who are unequal to the worries and cares of a large household. Women often begin and go on with eau-de-Cologne for a long time before they take any other intoxicants, but usually advance sooner or later, unless they abandon the use of alcohol altogether, to spirituous beverages. The essential oils which it contains are said to add to the alcoholic mischief on the organism.

Lavender essences are also used for purpose of alcoholic indulgence, though as yet only to a comparatively trifling extent, and are generally drunk when ordinary inebriant drinks are not available.

MORPHINISM.

Morphinism and opiumism present characteristics somewhat different from alcoholism, though morphine, opium, and alcohol have the common property of establishing a hold on the organism, especially on the nervous system. They all have the power of creating an appetite for themselves which has an imperious tendency to dominate the life of their victim. Opium is a narcotic poison, acute poisoning by which is the cause of a considerable number of deaths, in England and Wales alone 171 having been reported as taking place in 1891. When an excessive dose is taken by a novice the first sensation is of pleasant exhilaration, which is succeeded, usually after a brief space, by a weary, languid, heavy oppressed feeling of drowsiness with an overpowering tendency to sleep, and an utter inability to do anything. At this stage of opiate intoxication the person may be momentarily aroused by a loud noise, but immediately again drops off to his stuperose sleep. Later, when coma with stertorous breathing supervenes, he cannot be roused. The pupil's are nearly always unduly dilated, though at first they may be temporarily contracted, and are insensible to light. Though often lit up with a warm glow to begin with, this color gradually dies out of the face, which assumes a blanched, ghastly, peaceful look, the ghastliness increased by the heaviness of the eyes and lividity of the lips. The pulse, at first small, rapid, and irregular, with hurried breathing and warm perspiring skin, as coma sets in becomes full and slow; the respiration, which is now stertorous, being also measured and slow, with pale and cold skin. Very rarely there is purging, oftener emesis which is a hopeful sign of recovery if it occur before absorption with the consequent stupor. One of the most characteristic symptoms is copious perspiration, even though the skin is cold to the touch. The other secretions are generally arrested. On recovery there are sickness, vomiting, cephalalgia, anorexia, and a feeling of indescribable languor and powerlessness. The temperature is raised in the early stage, but before death takes place is subnormal, never, however, sinking so low as in fatal cases of acute alcoholic poisoning. Generally before a fatal termination there are muscular flabbiness and relaxation of the sphincters, dropping of the lower jaw, a thready pulse, and pupillary insensibility to light. In some cases there is said to be a loud mucous rattle in breathing, with, especially in children, convulsions (Taylor and Stevenson). Usually the leading symptoms, with the opiate odor of the breath, are sufficient to point to opium, but contraction of the pupils is also seen in apoplexy (sometimes only of one, at other times of both pupils, as in apoplexy of the pons), and to a less degree in poisoning by carbolic acid, the latter being discriminated by the odor and by the condition of the mouth. The greatest difficulty is experienced in the comatose stage common to all these three conditions, as well as to others. From forty to fifty minutes is probably the average interval before symptoms of poisoning show themselves, but the writer has seen a patient drowsy and stupid in eleven minutes, while occasionally hours (fourteen hours in one case) have been known to elapse. Generally the poison is more rapid in effect when swallowed in a fluid form, still more so when injected under the skin. On post-mortem examination the most general appearance, not always present, has been vascular congestion of the brain and its membrane. Effusion into the ventricles has mostly been absent. Sometimes there has been a darkish hue of the blood and even of the gastric mucous membrane. On the whole, appearances distinctive of opium poisoning have been absent. Stevenson has known three-fourths of a grain of solid opium kill an aged woman. On the other hand the writer has known recovery from very large doses when vomiting occurred soon, either spontaneously or induced. Opium is specially fatal to children. An infant seven days old died in eighteen hours from one minim of laudanum. According to Taylor and Stevenson, in fatal cases death usually takes place in from six to twelve hours, but has occurred within an hour.

Morphine being the principal alkaloid of opium, what has just been stated of opium is generally applicable to this constituent. The symptoms may manifest themselves in two to three minutes when the drug has been taken hypodermically, and in four to five minutes after it has been swallowed in solution. Congestion of the face and hands with a greater tendency to convulsions (though these are often absent) are superadded to the ordinary symptoms of poisoning by opium. The pulse is more uncertain and irregular in acute morphinism. One grain of hydrochlorate of morphine, administered by mouth and under the skin, has killed a healthy adult. The post-mortem appearances are similar to those after opium poisoning. Traces of opium and morphine have only occasionally been found.

In the chronic opiumism and morphinism of opiomaniacs and morphinomaniacs these habitual users gradually accustom themselves to enormous doses without any apparent immediate lethal effect. The writer has known a pint of laudanum to be taken by an adult female daily; and in the same time one hundred and fifty grains of solid opium in divided doses of thirty grains each by a male. In another male case sixty grains of hydrochlorate of morphine were injected daily. These, however, are exceptional cases in which the indulgence has been extended over a term of years, the quantity having been very gradually increased. The bulk of chronic opium and morphine devotees have advanced to the following daily quantities on an average: tincture of opium fully an ounce, opium about thirty grains, morphine salts about eight grains. A single woman of thirty regularly drank one and one-half ounces of laudanum, frequently, for a spell of a week or more taking up to three ounces every day. The wife of a medical man, forty-five years of age, for seven months drank one and three-quarter ounces daily, equivalent to the soluble parts of fifty-seven grains of solid opium. A married lady, aged forty-six, drank daily seven ounces of a mixture composed of equal parts of laudanum, spirit of lavender and spirit of chloroform. An adult aged fifty, for years consumed every day two and one-half ounces of laudanum and one and one-half ounces of paregoric.

The *pathology* of opium presents differences from that of alcohol. In chronic alcoholism the post-mortem appearances are significant of profound and widespread pathological inflammatory changes, of structure-destruction, tissue-degradation, and connective-tissue proliferation. In chronic opiumism usually the textural destruction and tissue inflammatory degenerations are practically absent, while the disproportionate fibrous-tissue redundancy is comparatively slight. The hyperæmic condition of the brain, lungs, liver, kidneys, and bowels, and occasional extravasations of blood sometimes described as having been present, with at times inflamed or suppurating lymphatic glands, are usually traceable to morbid affections apart from and concurrent with morphinism, though the cerebral anæmia is a sign of opiate tissue-starvation. The paleness of the heart muscle noted by Levinstein, with occasionally cardiac hypertrophy and fatty heart observed by Hirschfield in two cases, are also conditions arising from functional innutrition. The accounts of spinal cord granular degeneration have been too vague for acceptance. In alcoholism, too, there is frequently, especially in beer-drinkers, an excess of fat; in opiumism an entirely opposite condition—a complete absence of it. The shrunken and wasted vessels with the textural atrophy are a sure sign of innutrition and tissue-starvation, explaining and accounting for the shrunken and emaciated appearance of many opium inebriates. In a very small proportion of examinations by the writer traces of renal disease have been found, but the proportion is so small (probably not four per cent.) as to be fairly attributable to renal troubles existing prior to, or independent of, opium addiction. This remarkable difference between the post-mortem appearances of chronic alcoholism and those of morphinism is clinically represented during life, as there is no peripheral neuritis observed in pure morphinists. Other organic lesions which are ordinarily prominent on inspection of the bodies of chronic alcoholists are usually conspicuous by their absence from the dead bodies of opiumists; such are cirrhosis of the liver and kidneys, thickening or thinning and erosion of the gastric mucous membrane, and fatty heart. In addition to this difference in physical degradation, another striking distinction lies in the paralyzant action of alcohol. Opium and morphine may rarely be contributory factors in the induction of paresis, but they have no direct paralyzing influence on the nervous system. On the other hand alcohol is a paralyzant from first to last, its injurious march throughout being, in the expressive language of Lauder Brunton, one of progressive paralysis. As there are no post-mortem signs of irritation and inflammation and no corresponding inflammatory symptoms, as a rule, during life, in the person addicted to opium; so there is not in him generally the tendency to violence which distinguishes so many alcoholists. The charges of criminal violence brought before magistrates are accordingly few in opium-taking communities, in comparison with similar accusations in countries where alcoholic beverages are the general means whereby intoxication is attained. The same is true of mental diseases, inclusive of insanity, which are the product of brain irritation, disturbance, and degeneration. Both opium and alcohol by their narcotizing anæsthetic influence benumb the nervous capacity and deaden sensation, while undermining by starvation the vital powers and the intellectual faculties, with a consequent depraved moral sense. The narcotic hold on the nervous system is so much greater with opiumists that they are more difficult to cure than alcoholists. Yet, besides this atrophic physical wasting common in varying degree to both poisons, alcohol exerts a directly degenerative influence on mind and moral sense by the pathological action set up by alcoholic special affinity for the nervous fluid. Thus it is that while alcoholism is an organic and functional malady, morphinism (under which term opiumism may be included and *vice versa*) is practically a disease of function only. The functional derangements of alcohol and opium (and morphine) are somewhat opposite. In the main the early influence of alcohol on the generative function is inflammatory and excitant, especially in the male; while in the female alcoholism leads only indirectly to excitation and occasionally adversely to fertility by provoking menorrhagia, post-partum hemorrhage, and subinvolution of the uterus. In over sixty per cent. of the writer's cases of uterine hemorrhage of all origins except cancerous, alcoholic irritation has been the *fons et origo mali.* On the contrary, by its depressant influence on the general nervous system and on the special nerve-centres, opium is a common cause of impotence in men and of sterility in women, virility and reproductive capacity usually returning after the abandonment of the opiate narcotic. Vital temperature presents a characteristic difference in the operation of the two poisons, alcohol exhibiting a strong tendency to lowering, and opium to raising, the temperature of the living body. These general differences in the effects of these narcotics on human beings are to a considerable extent represented in transmissions to progeny. The influence of heredity is not nearly so observable in opiumists as in alcoholists. In the writer's experience there has been a family history of alcoholism or an allied neurosis in a little over one-half of the alcoholic cases, but only in less than one per cent. of the opium cases. This experience must, however, be subject to the important consideration that his practice has been principally among Anglo-Saxons, who have so recently taken to opium indulgence that there has not been time for opiate heredity to manifest itself, nearly all the cases having been the only examples in the family. But even in Eastern countries opiate transmission has not been scientifically recorded as having occurred in a large proportion of cases. There are apparent exceptions to this, but these may in great measure be accounted for by local exciting causes, such as malarial conditions, as over certain regions of a moist and swampy character the population have long been opium users. In England, in some of those localities which have been drained, the opium indulgence has vanished with the malarial troubles. Over many Eastern areas, also, opium has long been resorted to, to stay hunger-crave arising from the general starvation. A peculiar symptom common to alcohol and opium, often present where both substances are heavily indulged in, is *cardialgia.* This symptom is seen more frequently in periodic than in constant inebriates, setting in within a couple of days, or even sooner, of the beginning of an alcoholic paroxysm, and subsiding in a few days after complete abstention from the poison. Opiate cardialgia is usually seen in constant opiumists. In many cases with no history or other apparent symptom of excess, the writer has been enabled to diagnose alcoholism or opiumism. Opium devotees are, as a rule, regular users of the drug; alcoholic inebriates may be either periodical or constant in their excessive drinking. Opium, too, is mainly a *solitary* indulgence, frequenters of opium dens resorting thither not for the pleasure of opium eating or smoking in company, but simply because in these places they can readily procure their favored solace. Even when taking opium along with other opiumists they have little or no social intercourse, each indulger quietly enjoying his own gratification. With alcohol it is otherwise. Though some alcoholists drink to themselves and for themselves in solitude and secretly, the larger number drink (at least at the outset of their alcoholic career), with the social accompaniment of "good company," a large proportion of these latter never drinking alone. Thus while opium-takers are generally solitary, alcoholists are mostly social, though sometimes solitary. The aspect of the alcoholic grog-shop and the opium den clearly exhibits this difference. From the former sounds of mirth and converse, liveliness, and it may be strife are heard, the inmates generally being animated or noisy; from the latter rarely is there a sound heard, and if you penetrate within you will see, but not usually hear, pallid, taciturn persons silently reposing or asleep. The associated vices are different. Alcohol inflames the passions; hence lust, contention, and rage are often the issue, with an alarming consequent crop of blows and graver assaults. Opium, though occasionally in the fleeting preliminary stage of excitement it provokes to violence through resistance to acute narcotic lewdness, on the whole calms and soothes; yet it tends to nervous disturbance and excitement which often lead to speculation and gambling. Even on truthfulness alcohol has a more destructive influence than opium. In 98 of 100 cases of alcoholism the subject denies that he takes too much; in half the cases the opium-addicted admits and deplores his degrading enslavement. The complicating maladies are of differing types, though some affections such as dyspepsia and neuralgia are common complications. Alcohol is apt to induce hepatic, renal, respiratory, cerebral, and periphero-nervous affections, and dropsy or anasarca; opium gives rise to transient albuminuria, aguish attacks of pyrexia, shivering, trembling, delirium, dysentery, and emaciation. Alcoholism ordinarily has as it were a long, opium a very brief, incubation. While chronic alcoholism insensibly sets in, during a period extending over months, opiumism may be established in from a fortnight to a month. In both cases of toxication, a generous non-narcotic diet impairs the rapidity of the toxic process. A curious difference between alcohol and opium is that while all animated beings seem to be liable to be injuriously affected by the former, many animals are uninjured by the latter. From time immemorial certain Oriental races have been in the habit of making their war elephants drunk and therefore maddened so as to disorder in drunken fury the ranks of the enemy, a practice sometimes disastrous to their own forces, as in their blind fury the intoxicated elephants did not always discriminate between friend and foe. Pigs have been supplied with drink as a scientific experiment, with the result that they have died from both acute and chronic alcohol poisoning. Romanes experimented even on jelly-fish, finding that these delicate and beautiful creatures under the influence of alcohol tumbled over and acted erratically, as clearly intoxicated as the staggering, stumbling drunken men and women so frequently picked up drunk on the streets of some modern civilized cities. Contrariwise many animals live and thrive on opium, markedly goats and pigeons. This difference in behavior of alcohol and opium in the living bodies of the "lower" animals may partly be explained by the differing influence on vital temperature, opium slightly raising this as a rule, alcohol exerting so lowering an influence that in fatal cases the clinical thermometer may record a fall of from 3 to 9 degrees or more. The opiate rise in temperature does not last long, and as regards sensation inebriates in both opium and alcohol often have a feeling of peculiar chilliness. The extent of alcoholism and opiumism varies in different countries, as does the proportion of sex. In England, though opiumism is steadily increasing, there is probably less than a hundredth part of what there is in the United States, and there are more excessive users in the ratio of the whole population in Western than in Eastern peoples. Though "moderation" is more difficult with opium than with alcohol, it would be a mistake to concede what is often loudly proclaimed, that in India the majority of opium-takers are what is commonly called "immoderate" in their indulgence. On the contrary, the grossly intemperate users there are in a minority, by far the greater number limiting the amount of their daily consumption. In China the evils arising from opium are much greater than in India, the racial factor affecting the constitutional susceptibilities, though the climatogical conditions have an influence and the potency of the drug is greater. Further, the proportion of Anglo-Saxon opium-consumers who succeed in permanently restricting the quantity of the narcotic is much less than among Eastern communities, except in Burmah, the Burmese having shown so serious an extent of opiate mischief that the British have prohibited the sale of the drug in that country. Michaut (*Medical Age*, September 11th, 1894) states that the Annamese use opium without exceeding their regular daily dose, as do the majority of the Chinese. The European, on the contrary, is more liable to opiate intoxication partly because he exceeds the quantity adequate to yield intoxication, partly because he exhibits a greater susceptibility to opiate narcotization, the toxic potency being attenuated both in the Annamese and Chinese by heredity through successive impregnations of the individuals of the same family. As opium diminishes arterial tension it may produce passive congestion of the kidneys and other organs, but the writer, though always expecting to find albumin in the urine in such cases, has never found it except when albuminuria has preceded opiumism. Other observers have, however, met with post-opiate albuminuria. Atmaran, citing two cases of opium-eaters, each consuming over 240 grains daily, who suffered from albuminuria (*Indian Medical Record*, April, 1893), says opium smoked causes muscular debility. The muscular coats of the intestine and respiratory passages are affected, inducing chronic diarrhœa or dyspnœa. The paralysis of the muscular tissue of the stomach leads to diarrhœa, a grave malady in countries where dysentery is endemic and cholera frequent. In many chronic opium-smokers diminished accommodation, diplopia, and epiphora are the issue of opiate paralysis of the visual muscular apparatus. With injuries or acute inflammatory diseases in chronic excessive opiumists there may be a delirium somewhat similar to that liable to occur after sudden discontinuance of the drug. In heavy opium-smokers who exceed fifty pipes daily, or in those of from five to eight years' standing, extensor paralysis of the hands and fingers resembling that of saturnine origin occurs. This opiate paralysis is differentiated from that of alcoholic parentage by the former exhibiting no muscular atrophy and the sensibility remaining intact. The *modes of use* affect the rapidity with which the intoxication symptoms appear, and the degree of the action of the narcotic. Opium is more quickly absorbed when smoked than when eaten, but is then less injurious as only a comparatively small quantity can be inhaled in a given time. When eaten there is apt to be greater digestive disorder, and a more speedy and permanent establishment of opium-addiction. The effects of opium taken in a liquid form are manifested in pretty much the same time as when the drug is consumed in solid form. Though a fluid preparation is more speedily absorbed, this is counterbalanced by the smaller dose which can be swallowed at a time owing to the greater bulk. No mode of taking morphine is, however, so rapid in action as subcutaneous injection, the effects appearing almost immediately. This prompt operation, combined with the ease with which a syringe can be carried about and self-applied, have secured the popularity of hypodermic administration among morphine-takers. The dexterity and apparent unconsciousness with which a morphinomaniac can, while conversing with any one, administer to himself a hypodermic dose, usually in the arm, is marvellous. Though morphinomaniacs leave the face, head, neck, and hands untouched, nearly the whole of the rest of the surface of the body is often speckled with puncture marks, the thighs and legs being thus most abundantly tattooed next to the arms. The *initiation* of opium addiction is largely due to either medical prescription or self-administration of the drug for relief from pain or sleeplessness, the latter being by far the most frequent origin. As to the former the writer has known cases (which are becoming less frequent as the medical profession is becoming more alive to the risks involved in the practice), where injudicious medical advice has sent the patient back to his home armed with a hypodermic syringe for the injection of a morphine salt whenever pain was acute. More accurate knowledge of the perils of narcotic dalliance, with a higher sense of professional responsibility, will, by and by, it is to be expected, considerably dry up this source of inebriate replenishment. The resort to the drug in order to obtain physical ease or sleep is most frequent among medical men.

Mattison (1894) has had seventy per cent. of his opiate clientèle medical practitioners, the bulk being hypodermic morphine injectors. There are various reasons for this extraordinary share of opiumism and morphinism by medical men. The broken rest, frequent physical overwork, and mental overstrain, with the uncertainty of calls inseparable from a busy practitioner's life, lead to neurasthenic exhaustion, and are apt to induce acute physical and mental suffering in addition to a variety of harassing though it may be not immediately fatal maladies; among such is neuralgia in some of its most painful forms, such as of the head, the heart, and the stomach, so excruciating as to prostrate the sufferer and incapacitate him for the duties of his calling. It has been urged that the frequent contact with the poison tends to introduce physicians to opiate indulgence. This, the writer believes, operates in from two to three per cent. of cases, and in his experience, is paralleled by a somewhat similar proportion of such cases among druggists, who also are continually coming in contact with these potent substances. A certain proportion of physicians, especially of the younger, fall under the fascination of opiate indulgence while watching the effects of small doses on themselves. But the most common cause leading to a habit, in medical and in non-medical persons, is the light-hearted employment of narcotics to procure the immediate assuaging of pain. There is too little patience with pain both within and without the profession in these days. A little more endurance and patience would in a large number of instances save men and women from becoming slaves to narcotism. If such endurance were oftener the rule, it would frequently be found that the pains would prove evanescent, whereas the administration of opium, though soothing for the moment, frequently tends to promote a renewal of the anguish. Resistance to an impulse to secure immediate relief of suffering is in many cases the most effectual abolisher of the original suffering, which can often be remedied by tracing and removing the cause.

There is a group of habitually excessive opium and morphine users who ought not to be fairly classed as subjects of a morbid tendency to excess, viz., those persons who are suffering from acutely painful malignant disease, such as some forms of cancer. Not all of this group are impelled to indulgence by a strong impulse to or crave for opiate euphoria. A few are, but the mass of such narcotic consumers for a time at least consume the drug solely for the assuaging of their intolerable physical anguish, and unless for mitigation of pain never resort to any drug. A very few of these never become morphinomaniacs, and if the physical suffering cease give up taking a narcotic. In a majority of these really non-culpable users, however, there is unconsciously established the opium or morphine craze. Even when this is fortunately not the issue, the writer has found that on the whole opium-taking diminishes the capacity to bear pain and thus in the long run is detrimental to the sufferer. In one protracted case, in which hypodermic injection had been employed to lull the acute pain from a gastric ulcer, and had been repeated two thousand times, the sensibility to even slight pain became so delicate as actually to add greatly to the suffering. By injection of pure warm water under the skin, the substitution being unknown to the patient, the man's hyper-acuteness of sensibility was gradually lessened and his capacity of endurance markedly fortified, till, in the course of a few weeks, his suffering was diminished by at least three-fourths, and his existence made comparatively comfortable.

The cultured classes are those who yield the largest proportionate tale of opium habitués, outside of purely medical circles. Insomnia and neuralgic agony, the former preponderating, are the principal exciting causes. Usually endowed with a specially highly strung nervous system, neurotic literary and artistic persons are subject to excessive nerve exhaustion which has a strong tendency to disturb or prevent sleep, and sleepless nights incite the worn-out and exhausted sufferers to snatch repose, at any risk or unconscious of peril, by the easily applied hypodermic self-administration of morphine. So with the agonizing pains of neuralgic origin. On such a susceptible neurotic constitution the drug acts like a spark on gunpowder. In a fortnight, even in a week, not a few such neurotics have fallen under the dominion of the narcotic. The *extent* of opium and morphine addiction (*i.e.*, habitual excess) in Anglo-Saxon people is greater than is generally supposed. Crothers is of opinion that there are one hundred thousand opiumists in the United States; but the writer believes there are not much more than a fiftieth part of that number in the United Kingdom. Yet in Britain this form of inebriate excess is steadily increasing. On both sides of the Atlantic males contribute the largest number of opiumists and morphinists, but females are in strong form, accounting for probably from fifteen to thirty per cent. and even more in some localities.

In England there has also of late years been a gradual extension of laudanum-taking in excess by various classes of overwrought women following trades, such as milliners; the consumption is limited, however, by the fact that their scanty resources prevent them from purchasing as much as they long for. Of course, in some countries where the general population use opium the proportion of the sexes indulging in the drug is more nearly equal.

Chlorodyne is one of the composite preparations of morphine which is also advancing in favor with inebriates. This narcotic liquid article varies in composition. Generally, however, it is mainly composed of a morphine salt with dilute hydrocyanic acid, chloroform, with or without cannabis indica, and spirit of ether, in combination with treacle or syrup and oil of peppermint or tincture of capsicum. In the writer's experience there has usually been one grain of morphine salt (in most specimens the hydrochlorate) to the fluidounce, but Stevenson has found the average to be three grains. In *acute* chlorodyne poisoning contraction of the pupils is almost always seen, but the addition of atropine, which has been alleged by some to occur, may account for the unequal state of the pupils met with in many cases. In all cases seen by the writer there was contraction of both pupils, even in slight cases. Chlorodyne symptoms are very much those of opium and its constituents, though probably, owing to the anæsthetics with which it is associated, the action when the compound is swallowed (the only way in which it has been taken) is slightly more rapid than when pure opium or morphine is taken. Women form the greater number of the subjects. The wives of members of the learned professions, chiefly the clerical, have furnished most of the cases which the writer has seen. In one instance of this kind the wife cost her husband for chlorodyne £220 in some six years, and even after he stopped her credit for fifty miles round, she managed to elude observation and secure supplies from distant towns. Curious to relate, though chlorodyne is very expensive the writer has known the wives of coachmen excessive chlorodyne consumers; some would sell the very bedding to procure the compound if their husbands did not pay the bill, one such married chlorodynomaniac having, after pawning her husband's clothes and their family bedding, stolen his employer's rug which she pledged for money to purchase a supply of her favorite intoxicant. In London several similar cases have been brought before the stipendiary magistrates of recent years, through the unfortunate husband's desiring advice from the bench. The quantity taken is gradually increased, till in some cases the average amount consumed daily has ranged from four to five ounces. In most of the male cases seen by the writer the chlorodyne addiction owed its initiation to the drug compound having been resorted to in medicinal doses of from five to ten minims for the arrest of diarrhœa, over which morbid symptoms chlorodyne seems to have remarkable power. This therapeutic potency is so valuable that, for some ten years a quarter of a century ago, the writer was enabled to travel over a considerable part of the world by simply carrying a small bottle of chlorodyne in his pocket. The control over diarrhœal symptoms appears to be equally great whether the trouble is of nervous or phthisical origin. Though the narcotic power of chlorodyne is similar to that of morphine with alcohol, and while the compound seems in the bulk of persons who use it as a strictly therapeutic remedy to exert no appreciable tendency to excess, on many neurotic constitutions it may within a week or less obtain a grip on the individual which, unless the compound is resolutely discontinued, insensibly brings him or her under its complete dominion. The chloroform or ether seems with the alcohol in which the peppermint or capsicum, the prussic acid and morphine are dissolved, to promote the rapidity and firmness of the chlorodyne hold on the subject. In his experience the writer has seen a preponderance of female victims, these having generally resorted to chlorodyne for the relief of neuralgia or other pain.

In chlorodyne intoxication there is a very fleeting stage of pleasant excitement, succeeded by profound sleep and stupor, with a lesser tendency to coma than in acute poisoning by either alcohol or opium. The *arrière goût* is characterized by intense and persistent thirst, and by a peculiar hot, burning, dry sensation localized in the pit of the stomach. In most cases chlorodynomaniacs are and have been addicted to alcohol, the writer having known only two individuals who were abstainers from alcoholic intoxicants.

The *course* and *termination* of a career of *chronic* opiumism (*chronic* poisoning by opium, morphine, chlorodyne, or other compound in which either narcotic predominates), unless arrested by either sudden or gradual abstinence, are on the whole typical. The chronic morphinist (he or she who has become a slave to the drug), partly because an increasingly larger quantity is required to produce the same effect as was at the beginning secured by a small dose, and partly because there is begotten a morbid accession of craving for a fuller, even if evanescent sense of blissful satisfaction, insensibly falls deeper and deeper under the narcotic dominion till there comes a time when the capacity for even this fleeting solace is utterly lost. Meanwhile much suffering has usually been endured, though in a few cases there is none. The sufferer becomes more and more subject to functional disturbances, bodily weakness, and mental indolence, the whole frame laboring under the slightest exertion. He is harassed by frequent palpitation of the heart, oppressed breathing, cramps in the abdomen and leg muscles, nocturnal pains, fitful sleep with terrifying dreams, trembling and fears at imaginary or real voices. There is an exaggerated sensibility, slight pains seem to be acute agony, resolution is transformed into irresolution, with uncertainty of purpose, confusion of thought, morbid melancholy and despair, anorexia alternating with fitful voracious appetite, general constipation with, it may be, frequent prostrating diarrhœal or dysenteric attacks, salivation, listlessness, and indifference to cleanliness, personal appearance, and the claims of duty. The moral sense is by and by perverted, so that the person's word cannot be relied on, and the no longer pleasant though necessary opiate oblivion is procured, if it cannot be honestly, by theft, the sale of one's living body, or murder. The morbid action on the reflexes is variable, those at an early stage being heightened and at a late stage diminished. It has been asserted that there is a special form of opiate peripheral paralysis analogous to alcoholic polyneuritis, but the writer has never seen paretic symptoms at all resembling those due to alcohol. Sexual function is in general disturbed. In the female amenorrhœa prevails, in the male impotence, but in both sexes functional normality is gradually recovered on abandonment of the drug. The effects of the cachectic marasmus, which is apt to have a fatal ending, often remain long after abandonment, though in most cases they are in time overcome. Death may supervene in various ways: from some intercurrent malady, opiumists being peculiarly prone to be attacked by some diseases, while apparently almost proof against others; from an overdose taken either intentionally or accidentally; or, at rare times, from the effects of the shock incident on some surgical operation.

The *treatment* has been a subject of considerable controversy. Levenstein ("Morbid Craving for Morphine," 1879) advocates the abrupt method of that entire abstinence from opiates, on which alone the opiist's permanent cure necessarily depends. It is just as necessary to secure abstention from the poison in chronic opiate as in chronic alcoholic poisoning, the difference lying in the mode of adopting an abstinent course. With alcohol there is a consensus of skilled opinion that the best and safest procedure is by immediate withdrawal. With opium and its preparations the indescribable suffering undergone in most cases complicates the remedial process. These symptoms are grouped by Levenstein under the general title of "abstinence phenomena," but in the writer's opinion mistakenly, as many of these symptoms are not experienced when the reduction of the daily donation is gradual; so that a better designation would be "phenomena of sudden abstinence." Yet in immediate and total withdrawal of the narcotic, all or most of these symptoms are not invariably present. Under both sudden and gradual discontinuance there is always a strong, almost irresistible craving for, or impulse to, opiate intoxication. Whether in the earlier stages of opiumism when the subject derives pleasure from a fresh dose, or in the later stages when there is only an overpowering necessity with no hope of pleasure but simply the momentary relief from an intolerable and insatiable morbid physical appetite, an imperious impulsion is in all cases felt, this being slightly weaker in the weaning process. In the drastic abrupt withdrawal, however, the agonies of the sufferer are so practically unbearable as a rule, that only in rare cases has the writer carried out this plan successfully. At first the patient is consumed with fear at the risk which seems to his distorted imagination to be imminent; then he implores or beseeches his friends and attendants to give him a dose; then he may be irritable, cantankerous, violent, and after crying out, finally moaning, and a prey to melancholic despair which at times generates suicidal impulses, or incites to crime. All this may be but a prelude, if opium is withheld, to rigors, nausea, distressing vomiting, excessive diarrhœa, insomnia, tremors, restlessness, parching thirst, exhaustive sweatings, chilliness, photophobia, wasting delirium, convulsive seizures, paralysis, collapse, and death. Minor troubles are difficulty of speech and articulation, languor, yawning, sneezing, coughing, palpitations, acute neuralgic pains, and various hallucinations. Whether all or only some of these symptoms are present, the subject is tortured by a prolonged intolerable agony. Under such conditions he is so infuriated and uncontrollable that he has to be secluded in a padded room, or forcibly restrained by attendants or nurses. These tortures may be to a slight extent diminished by certain measures such as a wet pack, but even then they are as terrifying as they are exhausting. This torturing and perilous system has been advocated extensively by various members of the medical profession, but in the writer's opinion is unjustifiable except in the comparatively few cases in which (mainly from the short term of addiction) there is little suffering. In one case, for example, of female chlorodynomania where a fluid ounce had latterly been consumed daily, and the whole term of indulgence had been only between four and five months, the compound was immediately and wholly withheld. In the forty-eight hours of nausea and sickness, during which time water with milk and effervescent drinks were administered, the suffering and depression were not more serious than from sea sickness for that length of time. No narcotic nor anæsthetic nor alcohol was given, and the patient on the third day could take arrowroot and milk and oatmeal gruel, on the fifth day ordinary food. Such cases are exceptional; but in the writer's experience they occur occasionally and should always be subjected to an immediate and total cutting off of the drug supply. Christison followed this method in all cases, but in his time morphinism had not arrived to the perfection of a "fine art." Erlenmeyer's plan of rapid reduction is productive of less risk, less suffering, and is as efficacious as sudden withdrawal, though even in professional circles there is a belief that the latter method is more effectual in preventing relapse, an erroneous tradition which in the writer's opinion has no warrant from facts. This method of rapid reduction, though as useful and much less perilous as well as less painful than the sudden withdrawal, is not wholly satisfactory. It involves a substantial amount of suffering which is in great part unnecessary. The gradual diminution of the daily allowance by an average quantity, allowing the final withdrawal to take place in a week or so, involves a condition of depression, of nervous distress, and of bodily disturbance from diarrhœal and other physical disturbance, which calls for alcoholic stimulants or substitutionary narcotics from which latter in their turn the patient must be gradually tapered off. The writer has found it advantageous to spread the reduction period over a term of from two to three weeks or longer, and he prefers to begin at once by administering one-half of the usual quantity taken by the patient just before the treatment is begun. The reduction period varies with the quantity habitually consumed, the previous duration of the indulgence, the idiosyncrasies of the patient, and his condition at the time. Each case needs to be separately studied, and to be treated according to its special requirements. During this gradual withdrawing process the writer finds valuable aid in the administration of various remedies and processes. The greatest benefit has been gained from moderate doses of the bromides in combination with a vegetable bitter and a narcotic, all in moderate doses. To allay the extreme nervous irritability a night draught composed of 20 grains each of potassium and sodium bromides, 15 minims each of tincture of cannabis indica and of hyoscyamus, and 20 minims each of aromatic spirit of ammonia and compound tincture of cardamoms, either in 5 to 6 ounces of water, or in about half that quantity of water with a copious draught of water immediately after the mixture has been taken. Five minims of tincture of nux vomica may be added if desirable. If the nausea and sickness, as is often the case, are severe and persistent, the writer prefers to prescribe the remedies in an effervescent form as generally more calmative to the stomach, more easily retained, and more grateful to the palate. He sometimes gives as a preliminary dose 4 grains of blue pill, followed by a seidlitz powder or a black draught next morning, or euonymin with calomel may be substituted for the blue mass. During the stage of gastric disturbance iced milk with lime- or soda- or seltzer-water, varied with malted foods, peptonized foods, albuminoid beef preparations, broths, plain soups, or farinaceous diet, will probably be found best adapted to the patient's condition. As the acute gastric distress abates, white fish such as sole, whiting, cod, plaice, or haddock (with or without a suspicion of lemon), or oysters, may be ventured on, the dietary being gradually extended till all ordinary simply cooked food can be thoroughly digested and assimilated. As soon as fatty food can be well borne, it is useful to counteract the effects of the prior nerve starvation. Next to the Romano-Turkish bath, and often even in preference to this, apart from the fact that this fascinating and elegant article of modern luxury is usually not available in a private household, there is nothing so soothing to the disturbed and irritable nervous system as a hot or even cool wet pack such as has already been described when treating of alcoholism. In morphinism and addiction to opium and all allied substances, alcohol is contra-indicated. The administration of this inflammatory and exciting narcotic adds only "fuel to the fire." Alcohol, too, being a depressant, after its evanescent excitant effects, deepens the depression experienced by alcoholists and opiumists, and should be resolutely withheld. Coca has been highly lauded; but the writer has seen no benefit from it. In his view narcotic substitutes as a rule are a mistake when taken for this purpose, alcohol almost never being useful, cannabis, cocaine, and chloral only when given in limited doses. At times once, twice, or thrice during the curative process, it may be desirable to give an ordinary full medicinal dose of opium; but in many cases no such necessity arises. Lavage of the stomach is sometimes useful. Bathing, especially hot, should be encouraged, and exercise gradually fostered. One thing is indispensable, except in exceedingly rare cases, when the patient has a strong will and can himself carry out implicitly the directions of the physician; that thing is complete control over the patient. This should be continued from first to last, till the convalescent can be trusted out alone. The writer has a strong conviction that the short term of a few weeks, so frequently considered sufficient to secure all the advantages of remedial treatment, is quite inadequate. In his observation only a small proportion of permanent cures is effected in this way, though undoubtedly, especially in strong-willed persons, six to nine weeks have afforded that opportunity of starting afresh without opium which is practically all that is needed in certain instances. The limited time available to many opiumists, with the expense involved in prolonged residential care under special remedial conditions, which in nine-tenths of all cases is by far the best procedure, complicates the position in the case of inebriates who have exhausted their resources in purchasing so much of an expensive drug and who have families dependent on them. This difficulty is met only in one special institution (founded by Mattison in Brooklyn), where some free beds are provided; but it is to be hoped that municipal or governmental authorities will, both for opiumists and alcoholists, supply the necessary provision. The brain and nervous system should be built up anew, and the replenishment of sound tissue is not a speedy process. The writer thinks that three months ought to be the minimum term of residence to be aimed at, but that wherever this is possible, at least double that time should be allowed. Alcoholism, as a rule, calls for a minimum detention of twelve months, but opiumism does not demand, unless in certain cases, so lengthened a term. At the same time only good can be the issue of as long a period in opiate cases, if the patients can be persuaded to occupy their minds with suitable occupation, and strictly follow the life and regimen laid down for them. It will be found that the length of time needed to return the best result varies with the individual patients. While for some three months would suffice, others would require six, eight, or twelve months, or even longer. The greatest danger lies in the over-confidence begotten of the wondrous elasticity of spirits usually experienced on emerging from under the depressant influence of the narcotic. Various drugs are helpful at different stages of treatment. While narcotics are useless and risky if employed as substitutes, they may be valuable at times if prescribed only in strictly therapeutic doses to meet certain indications. Among the most serviceable drugs are strychnine especially in the form of nux vomica, quinine, iron, belladonna, digitalis, vegetable bitters, alkaline draughts, diuretics, an occasional draught of Epsom or Glauber's salts, Rubinat, Hunyadi János, Friedrichshall, or other similar mineral water, which often acts like a charm in clearing the primæ viæ and lightening the general malaise and feeling of oppression. Massage, electricity, and bathing, at first warm, afterward cold, are invaluable. In the convalescent period, which ought to be spread over several months, the will should be strengthened by regular exercise in the open air, muscular exertion at some mechanical work indoors, administration of the phosphates or phosphites, of quinine or other tonics, the elevating influences of music and the fine arts, intellectual culture, moral and religious considerations. Along with all this, the patient should be plainly instructed in the physiological and pathological effects of narcotics and anæsthetics on the living human body, abstention from all such perilous substances being clearly laid down as a lifelong essential of permanent cure.

Sparteine and nitroglycerin (the former hypodermically in the form of the sulphate, in doses of from 2 to 4 centigrammes, the latter gr. $\frac{1}{100}$ in tablets, or in a one-per-cent. alcoholic solution) are recommended by Ball and Jennings as "heart stimulants;" but the writer has found digitalis and strophanthus quite satisfactory. To Mattison in 1876 belongs the credit of the first promulgation of the rapid method of reduction of the amount of opium in from six to ten days. To him also we owe the preliminary sedative process, by which he claims to have attained a maximum sedative influence at the time of maximum nervous irritation from the opium-decrease and quitting. In suitable cases, Mattison, while recognizing that the bromides are contra-indicated in certain cases, gives sodium bromide in initial doses of 30 grains, twice daily, at 10 A.M. and 10 P.M., increasing the dose 10 grains each day until the maximum of 100 grains semi-daily is reached on the eighth day. On the ninth and tenth days, this maximum 100-grains dose is given in the evening only. He then meets any reflex symptoms by codeine, which is rarely called for till after the complete discontinuance of the opiate. A favorite solution is made of the sulphate in boiling water, two grains to the drachm. The dose is 1 to 3 grains, by mouth or skin, every two to four hours, in gradually decreasing dose and increasing interval till no longer required. Codeine in this disease was first suggested by Lindenburger of California in 1885. Mattison prefers trional as a hypnotic, the usual dose being 40 grains for males and 30 grains for females. It is given for the first six or eight nights decreasing in quantity gradually to half the initial dose; thereafter, if necessary, chloral, paraldehyde, or cannabis being used. Waugh (*Quarterly Journal of Inebriety*, October, 1894) states that he has met with the best success in replacing morphine by eserine or physostigmine salicylate, which contracts the pupil, acts as a sedative, and raises arterial tension while slowing the pulse, but is a tonic to the muscular fibres of the intestinal canal and to the heart. He reports this remedy as producing a sense of comfort or euphoria equal or even superior to that of morphine. He administers $\frac{1}{100}$ grain hypodermically.

In all such cases, under present conditions the first prospect of cure lies in the sufferer's desire to be cured. If, however, reception and detention in a genuine home were involuntary for a period of from six to twelve months and more, the compulsory speedy or gradual, but thorough, withdrawal of all narcotics would tend to emancipate the opiumist from the power of the narcotizing agent, so that on an unclouded brain and undimmed intelligence such elevating and ennobling impressions might be made by appropriate teaching and counsel as would inspire the former listless and despairing narcotic slave with new and noble resolves. In this way there is good reason for the anticipation that a considerable number of even such involuntary *détenus* would be restored to the fulfilment of their duties in life. Reviewing the whole subject of treatment, the writer's opinion is that there is no specific for morphinism, and that patience, with a careful study of the peculiarities and indications of each case, will point out the line of treatment best adapted to the case in hand. He is supported in this view by the opinion of Crothers, who writes that he succeeds better with baths, Turkish and hot shower, than with any other remedies, though he tried all the new remedies in the rapid succession of their promulgation. Other preparations of opium have been employed with a view to narcotism, but little could be added for practical usefulness beyond what has just been stated.

CANNABINISM.

Though frequently seen in some Eastern countries, this habit is rare in Western communities. Peterson gives an account of the extent and effects of the inordinate consumption of bhang or hashish. On a visit to the lunatic asylum at Cairo, he found that 60 men and 4 or 5 women out of 248 patients had been recorded as having been indebted to hashish for their insanity. The drug, which has been in use in Egypt for six hundred and fifty years, is generally inhaled by smoking, and is sold in square and diamond-shaped lozenges of a black extract. Many cases end in chronic insanity, dementia, or death. The chief symptoms are indigestion and diarrhœa, dilated pupils, drooping of the eyelids, anorexia, and general debility. Mental disorder is ushered in by gradually increasing timidity, amounting at times to a *folie du doute* or agoraphobia. Gunja is stated by Pringle ("Indian Hemp from a Public Health Point of View," London, 1893) to be prepared from the dried flowers of the cultivated female hemp plant in Lower Bengal, under government license and supervision. In all the admissions where the cause of the disease could be ascertained, the percentage of the cases of lunacy declared to be due to gunja during two years prior to 1894 was, in Bengal, fifty-three per cent. When added to alcoholic intoxicants, as is sometimes the case in India, mad drunkenness characterized by homicidal violence and fatal foolhardiness is the result. Richardson believes that Indian hemp is older than wine as an intoxicant, that it was the "nepenthes" of Homer. Hashish is drunk, eaten, and smoked (the last with difficulty).

CHLORALISM.

This is a modern form of poisoning, chloral having been discovered only about thirty years ago by Liebreich. There have been many deaths from acute chloral poisoning, some of which have been sudden, while most of those who have died have never regained consciousness after once falling asleep under the influence of the drug. While a dose of 3 grains has been known to kill an infant of one year and 30 grains to cause the death of persons between twenty and thirty, recovery has taken place after the ingestion of 120 or even 460 grains. Owing to the uncertainty of the risks this narcotic should be prescribed only with extreme caution in doses not exceeding 20 grains or so if to be taken at once. If spread over a day the dose should not be more than 5 grains every four hours, for an adult. The action is uncertain, though the tendency is to death by coma. It is also believed that sudden paralysis of the heart is generally the immediate cause of death, which may also occur from arrest of respiration. In some cases of recovery there has been first flushing of the face, then vertigo with a sense of suffocation succeeded by a deep drowsy feeling. The chief symptom is the gentle and apparently natural sleep into which the chloralized speedily falls, during which (till the comatose stage has set thoroughly in, and even for a time in this state), as in acute opium poisoning, the sleeper can be roused only to fall asleep again almost immediately. As in acute alcohol poisoning, the temperature decreases. As deep coma advances the rate of the respirations is slowed, and though they may continue they become less full and more shallow, with muscular relaxation; the erewhile contracted pupils are now dilated, the temperature continues to fall, the pulse becomes either rapid or slow and irregular, getting gradually weak and thready, until in fatal cases the face appears flushed or livid or bloated and collapse sets in, followed by death. In these fatal cases the peculiar dry sweetish burning sensation, generally experienced in the œsophagus, extending upward from the stomach through the throat and mouth in chronic choralism, is sometimes intensified, though it does not generally amount to acute gastritis. Reflex action and sensibility are lessened or lost. In prolonged cases there may be an urticarial skin eruption.

The differential diagnosis is from the coma of other anæsthetic and paralyzing substances, such as alcohol and opium, poisoning by carbolic acid, and congestion of the brain. Carbolic acid is excluded by the absence of the characteristic dark greenish or black urine, of stains about the mouth and lips and charring internally, and of the carbolic odor of the breath. Opium shows greater and more persistent pupillary contraction, alcohol less, while the odor of each may be detected in the vomit, and emesis is more difficult of accomplishment in chloral cases. Cerebral congestion shows more unevenness of the pupils.

Treatment of acute poisoning should always be begun by emptying the stomach by a siphon tube or stomach-pump where it is too late to give a full emetic. As in poisoning by alcohol the surrounding temperature must be kept up and the external surface of the body enveloped with warm wrapping and hot applications, useful forms of which are hot flannels, and tin and india-rubber bottles filled with hot water. The patient must be kept awake by flapping the skin with a wet towel, flying sinapisms, sharp and loud calls, etc. A pint of hot, strong coffee should be given by the mouth, or, if that is impracticable, injected into the rectum. As in acute alcoholism, $\frac{1}{25}$ grain of strychnine nitrate might be hypodermically injected if 10 to 15 minims of tincture of nux vomica cannot be swallowed, and repeated occasional inhalations of amyl nitrite, with prolonged artificial respiration, are also useful in some cases. The application of a battery to the limbs may also be called for.

The *after-effects* which are observed in some continuous chloralists are generally unpleasant. There may be digestive disturbance, anorexia, with inability to go to sleep unless after a fresh dose. The circulatory system is slowed, the face assumes a bluish-purple aspect from the deficient aeration of the blood, the heart is laboring, and the respiration is oppressed. Lessening of the inhibition of the various nerve-centres is reflected in muscular unsteadiness. Vaso-motor disturbance is evidenced in the coldness and blueness of the extremities. Withal there is an overpowering listlessness, with an utter lack of energy. Especially in subjects of old age there is apt to be a disposition to cardiac asthma and syncope. On post-mortem examination, the chief appearance is usually a fulness of the vessels of the brain and also sometimes of the lungs, the blood being darker in hue as in asphyxia and correspondent to the dark-blue purple seen in living chloralists. The system may become so accustomed to chloral that the dose can be gradually increased to, as in one case, 300 grains daily in divided doses of 150 grains each, the individual having in nineteen months insensibly advanced to this amount from 15 grains once a day at bedtime. In addition to the functional disturbances already alluded to with the tendency to cardiac paralysis, the predominant peril threatening the chloralist is the risk of a fatal overdose. In one case, in a man aged fifty-two, of continuous alcoholism of several years' standing two doses of 50 grains each, the second an hour after the first, proved fatal. In another case, a young barrister, aged thirty-three, was found dead the next morning after having taken an accidental overdose, hundreds of gradually increasing doses having been taken prior to the last, with a lavish drinking of spirits at night. In these and other similar cases all the circumstances went to dispel any suspicion of suicide, though in a few cases there have been indications of a suicidal intent. Alcohol is peculiarly dangerous to the chloralist. The chloral-begotten tendency to cardiac debility and paresis is aggravated by alcohol, the deleterious action of the latter poison on the heart muscle being productive of fatty degeneration weakening the organ itself, while the alcoholic paralyzing influence renders the heart less capable of withstanding the paretic influence of chloral. Both poisons, too, tend to embarrass the circulation by imperfect aeration of the blood. Thus death frequently results from the combined action of chloral and alcohol, as in the instance just referred to. The writer therefore strongly urges the withholding of alcohol in all cases of chloralism, especially of acute chloral poisoning. Chloral is decomposed within the blood, giving off the chloroform which causes the sleep. For a time this new mode of narcotism advanced in popularity, but this advance in public favor was arrested a few years ago by attention having been drawn to the fatal risks of this form of indulgence. Now, however, the writer sees symptoms of a revival of chloral addiction. The sale of elegant preparations containing chloral, such as a syrup which is directed to be taken in teaspoonful doses (one such preparation contains half as much again in a teaspoonful as the writer would dare to give for a dose), without any reference to the character of the preparation, is a practice which cannot be too strongly condemned. Stevenson states that one patent medicine contains 22 grains in one fluidrachm. The composition of any patent or commercial pharmaceutical compound ought to be plainly set forth on the label, and special attention, by thick conspicuous lettering of a certain color, should at once arrest attention to the fact if the bottle contain any poison. Cases of death by misadventure are occurring frequently in Britain from laxity in this matter. Chloralism is practically never a social indulgence, but is a purely personal and private one. Chloral-takers consist mainly of littérateurs, professional men, and women of a highly strung and refined nervous temperament or high intellectual attainments, though a few artisans and artisans' wives have also been in the chloralist ranks. Insomnia from mental overstrain has usually been the occasion of the beginning of addiction, neurasthenic exhaustion, and irritability from domestic worries and a desire for relief from neuralgia and other pains, also increasing largely the number of habitual chloral users. Chloralism has generally been a further development of alcoholism, about four per cent. of the alcoholists seen by the writer having been chloral-takers. He has seen no abstainer from intoxicants in the person of a chloralist, though he knows abstainers who take a small dose of chloral at occasional times to secure a night's sleep, or the assuaging of acute pain.

The *treatment* of chronic chloral poisoning is on the same lines as those of chronic alcoholic poisoning, but the writer advises a rapid reduction spread over from two to five days instead of immediate withdrawal of the anæsthetic, with a plentiful supply of hot coffee, soup, and milk to begin with, rest in bed for a week with external warmth and a warm atmosphere, followed by a nourishing dietary and nerve tonics. Digitalis or strophanthus in small doses is also of advantage when combined with nerve tonics, such as nux vomica or strychnine in pill with oxide or valerianate of zinc, and extract of gentian.

CHLOROFORMISM.

The addiction to chloroform naturally falls to be considered alongside of chloralism, as the symptoms of the latter, especially in acute poisoning, are mainly produced by the action of the chloroform set free in the blood. Chloroform is a colorless fluid with a specific gravity one and a half times that of water, in which latter fluid it consequently sinks to the bottom. The chloroform of the British Pharmacopœia has a specific gravity of 1.497, and contains 0.2 per cent. of alcohol. Chloroform may be taken either when swallowed in liquid form by the mouth, or when its vapor is inhaled. Its action on the living organism is similar in kind to alcohol, but it is not so potent an irritant to tissue, or so permanently injurious to nerve structure.

When *drank* it is not so rapid or so powerful in its action as when inhaled as a vapor. It is taken as a liquid generally with suicidal design, but occasionally by accident. The symptoms are not so rapidly manifested as when the poison is inhaled as a vapor. Various persons have been able to move about doing little things, and even walking for a time before being suddenly overpowered and falling down insensible, after sometimes staggering for a few seconds, the unconsciousness quickly deepening into coma with complete anæsthesia, with stertorous breathing, dilated pupils, cold skin, imperceptible pulse, and occasionally general convulsions. Sometimes there is a facial flush, with emesis within from eight to fifteen minutes after the ingestion of the fluid. This vomiting sometimes retards or lessens, sometimes does not appear to delay or weaken in the least, the action of the anæsthetic poison. The temperature is lowered and the respiration slowed down. The smallest fatal dose recorded was one fluidrachm, which killed a boy of four years. There have been several recoveries of adults, after the swallowing of two ounces. One and a half fluid-ounces caused the death of a male alcoholic inebriate otherwise healthy, aged fifty-eight. The smell of the breath while alive, and of the fluid often found in the stomach after death, points to acute chloroform poisoning. As in other narcotic intoxications by poisons swallowed, the initial step in treatment is the clearing out of the chloroform left in the stomach by a full emetic of half an ounce of mustard or of wine of ipecac in warm or tepid water, or 20 grains of zinc sulphate, or 30 grains of powdered ipecac, given by the mouth or by the stomach tube; or apomorphine hydrochlorate, gr. $\frac{1}{10}$ hypodermically or gr. $\frac{1}{5}$ by the mouth. After vomiting, a solution of bicarbonate of soda in water should be given in large quantities. The patient should be aroused by wet towel flicking, mustard to the calves of the legs and over the heart region. A pint of hot strong coffee should be thrown into the rectum and nitrite of amyl inhaled frequently. Hot external applications are also useful. Special precaution should be taken to watch the patient's after-conditions; a fatal relapse has occurred in cases which bade fair to do well.

When *inhaled* undiluted, chloroform vapor acts much more promptly, especially when a glass of spirits has been given to a drinker immediately beforehand. Even when administered diluted with air, to induce anæsthesia for the performance of a surgical operation, death has taken place in less than a couple of minutes from the inhalation of half a fluidrachm and even less, though this has been very exceptional. On the other hand the writer has kept a woman with post-partum eclampsia continuously under chloroform for seventy hours.

When poisonous symptoms arise after inhalation the *treatment* is to see that there is no obstruction in the mouth such as by artificial teeth, to pull out the tongue with the forceps and clear the mouth, to loosen the chest wrappings, and flap with a wet towel the face and chest, to let fresh air into the apartment by opening windows and doors, and to douche the chest and head with alternate hot and cold water applications. Artificial respiration should be begun at once, the body being inverted so that the head is on a lower level than the remainder of the body and rests on the floor. Nitrite of amyl should be inhaled from time to time. A battery might be applied in a weak interrupted current, for a few minutes only, one pole at the pit of the stomach, the other pole over the larynx. When the patient seems to be *in extremis* a couple or more violent blows on the chest quickly given may restore the action of the heart. Atropine hypodermically administered, in doses of gr. $\frac{1}{120}$ to gr. $\frac{1}{40}$, has been recommended.

Chronic chloroform poisoning is another illustration of the injury wrought on man through the poison indulged in, to which he is impelled by the narcomaniacal diathesis. Though the sensation of chloroform is to a novice by no means pleasant, from the pungency and smell of the fluid to the reactionary distress, this does not appear to present a barrier to renewed intoxication. Though the chloroform tippler knows the risks he runs through the possible passage from insensibility to death during the anæsthetic sleep, a disaster not uncommon, yet he cannot resist the morbid impulse to take the drug greedily. The symptoms are the same in kind as in acute chloroform poisoning, but a tolerance of the drug is developed by repetition, so that eventually large quantities can be borne without a fatal issue during the unconsciousness of a chloroform "drunk." There is, however, an occasional verdict of a coroner's jury of "found dead." The chronic toxication does not appear to produce any organic lesion, unless a weak flabby heart is considered to be such. The pathological after-death appearances are more indicative of functional disturbances. The most prominent symptom during life is gastric derangement. Though nausea is not felt at first after the initial small doses, as the dose is increased (which is usually rapidly done) the subject is harassed by nausea and emesis, dyspepsia, dry, somewhat burning thirst, præcordial pain, anorexia, and flatulence. Physical and mental depression soon sets in, with nervous trembling, languor, a lack of interest in everything around, drowsiness, sluggish circulation, and emaciation, with coldness of skin. The person gradually poisoned by chloroform assumes by degrees a more and more haggard aspect, looks weary, worn, dejected, half-starved, and hopeless. Physiologically chloroform first relaxes, afterward contracts the vessels. As the wretched and wasted victim descends on his chloroformic "down grade" to premature death, the danger of syncope and sudden death, especially during the intervals between the "bouts," which has always been present, increases *pari passu.* The subjective sensations of heat and cold are different from those characterizing chronic alcoholism. Both chloroform and alcohol rob the human economy of vital heat, but after alcohol the skin is warmer, while after chloroform the skin is colder. Alcohol is a nervous vascular relaxant, chloroform relaxing only temporarily and then contracting the capillaries, thus producing pallor, chills, and nausea. Medical practitioners form a large proportion of chloroform-takers, and the form of this indulgence is solitary. Chloroform inebriates are usually alcoholics. The writer has known only one abstainer from alcohol who took to chloroform, and he was afterward cured. Chronic chloroformism is both periodical and habitual, mostly the former at the outset, but gradually developing into the continuous form, the sober intervals contracting in duration. Inhalation has generally been the mode of taking by chloroformomaniacs. A married woman of forty-two years, who was found dead from syncope on May 7th, 1888, while insensible from chloroform, had been in the habit of consuming for at least ten years a pint daily, and pouring the chloroform on a blanket for inhalation. A male chloroform inebriate of fifty had long been accustomed to purchase daily two ounces of chloroform at a time from four different druggists, and had been observed to adjoin to a neighboring coffee-house and inhale the drug from a handkerchief over his face. After this practice had been followed for some years, he could rarely find a druggist who would sell him more than half an ounce at a time. Most of the cases are medical men or persons more or less associated with drugs, and the age ranges from thirty-five to fifty-five years.

ETHERISM.

Ether has a sweetish, pungent, hot taste, and when drunk gives rise to a burning feeling of constriction in the mouth, fauces, and throat. Its action is exciting, exhilarating, and intoxicating, very much like that of chloroform. Inhaled it is longer in rendering a person insensible and more of the anæsthetic is required for that purpose than is the case with chloroform; when swallowed, however, ether is more rapid in taking effect. Generally speaking, what has been stated of chloroform applies to ether. To Morton, of Boston, U. S. A., we are indebted for the introduction of ether to promote anæsthesia, in 1846. Ether purus, free from alcohol and water (sp. gr., British

Pharmacopœia, 0.720; United States, 0.725), though the most free from drawbacks, has, though in fewer instances than have occurred under chloroform, caused death by acute poisoning. Though its odor is more unpleasant to the patient, and though it is more costly from more of it being needed to put the subject into a deep enough anæsthetic sleep, and though it involves a little more time in inhalation, its greater safety, with the lesser discomfort from the more manageable emesis, have secured for it greater acceptance for surgical anæsthesia from surgeons generally.

Only one or two fatal cases are known to have resulted from ether when swallowed, and there is no history of the health of these victims.

The *treatment* is similar to that applicable to acute chloroform poisoning, with the addition that where tracheotomy is called for, this should be done at as early a stage as possible.

Chronic ether poisoning has, however, been exhibited in recent years on quite an extensive scale, especially in certain localities in Ireland. This country has presented two, both now historical, most remarkable series of psychological phenomena in relation to anæsthetics. In 1838 a simple, unostentatious Roman Catholic priest, Father Theobald Mathew, commenced a crusade against alcohol, and within three years had administered the teetotal pledge to millions of his countrymen. Though a remnant remains true to the pledge to this day, Ireland drinks as much proportionately to her diminished population as ever she did.

About 1842, to a few newly pledged abstainers a medical man gave a drachm of ether, which was not believed to be a violation of the pledge. The practice increased so rapidly that a village of 300 inhabitants had soon one shop for the sale of ether to every 23 of the population. In a few years ether-drinking had spread more or less over an area of nearly 295 square miles with a population of almost 79,000, including the mountainous districts of Derry and Tyrone, and partly of Armagh and Antrim. To a limited extent this form of intoxication extended to Dublin and elsewhere in Ireland, Glasgow in Scotland, Lincolnshire in England, London, Norway, and New York, and latterly France. In Ireland, though ether purus (B. P.) has been the variety taken by several of the cases seen by the writer elsewhere, the general preparation in use was sulphuric ether, containing eight per cent. of alcohol and water with ninety-two per cent. of ether purus, soluble in all proportions in alcohol but only in the proportion of 1 in 10 of water. The specific gravity ought to be (B. P.) 0.735; (U. S.) 0.750.

Sulphuric ether is a rapid, powerful, diffusible stimulating narcotic, anæsthetic and antispasmodic, and is a medicinal agent of great therapeutic value, with a strong, penetrating smell, sweetish, burning, and pungent to the palate. By the Act 18 and 19 Vict. c. 37, in 1855–56 spirit of wine was permitted to be used in arts and manufactures in Britain duty-free if mixed with at least one-ninth part of methylated alcohol (wood naphtha, spirit from the destructive distillation of wood); this measure was intended to prevent the use of the duty-free and therefore cheaper spirit as a beverage, by reason of the nauseousness of the mixture. This precaution was, however, nullified by the destruction of the fouling methyl ingredients during the conversion of the mixed spirit into ether; so that practically methylated ether, though so much cheaper (one-seventh) than ethylic ether (ether made from ethylic alcohol), is undistinguishable by the palate. The cheaper ether accordingly supplanted the dearer, the cheapness causing a greatly increased consumption. The intoxicant was brought to the doors of the people by hawkers, was sold in shebeens and groceries, being often exchanged in barter for eggs and other produce of the farm-yard. "Draughts" of nearly two teaspoonfuls were sold for a penny. In Ireland agricultural laborers, small farmers, and workmen formed the majority of the ether drinkers, the women nearly equal with the men. Cases occurred also in better off and more cultured circles. Children from ten to fourteen years of age were known to arrive at school with an odor of ether in their breath. The quantity taken at one draught ranged from a teaspoonful by a novice, to one or two or more fluidounces by a confirmed etherist. The average daily quantity swallowed by a "moderate" ether-drinker was often ¼ ounce three or four times in the twenty-four hours. In confirmed cases half a pint has been daily consumed by "seasoned casks." In England ether is generally inhaled, one pint per diem having been the daily allowance of the most advanced ether-tipplers. As ether is sparingly soluble in water (1 in 10) the attempt of a foreigner on Irish soil to attempt to drink the pungent ether in water often used to afford great amusement to the native population, though the accomplished and thoroughly trained etherist may in time arrive at the perfection of being able to toss off his ether "dram" at a gulp without even holding his nose. The process of ether-drinking is at the outset generally a comedy in two acts, after an overture in two parts. The overture, Part I. consists in washing the mouth with cold water; Part II. in drinking a draught of cold water, with the object of cooling the mouth and throat. In Act I. the ether is swallowed "neat;" in Act II. there is a second draught of cold water drank, to "keep the ether from rising." The onset and decadence of intoxication are so rapid that a man or woman may get drunk and sober again within from two to four hours. Ether drunkenness pure and simple may be described as effervescent and hysterical; combined with alcohol, violent and maniacal from a few minutes to half an hour. In one case, that of an educated man of thirty-eight, who had been an etherist for between six and seven years, there was immediate paternal alcoholic heredity, his father and two paternal uncles having died of alcoholism. The etherist himself was a strong opponent of alcoholic-drinking, and was under the belief that he had escaped the family legacy of narcomania because he abstained from alcoholic intoxicants. In another case, a medical man of forty-nine, who had first habitually indulged in alcohol and opium and had been an etherist for seven or eight years, the ether drinking had been originally irregularly periodical, and had gradually developed into continuous tippling. Most of the cases seen by the writer have been as continuous as occasion would allow.

Of the *pathology* of chronic etherism not much is known. In the one or two fatal cases in which a post-mortem examination has been made, no special appearance was noted.

The *symptoms* seem to be chiefly those of a purely functional disturbance. Acute and chronic gastritis is frequently observed, with digestive distress, epigastric burning pain, and anorexia. We find insomnia and troubled sleep, pallor, tremors till a fresh dose is taken, fear, gloom, despair, suspicions, nervous anxiety, and chills. The skin is lemon-colored or cyanosed, the heart-beat is intermittent, the reflexes are exaggerated, and there is a feeble tottering gait, with an increasing feeling of failing strength in the lower limbs. The chronic etherist, if he does not stop in his headlong career, has a tendency to become shrivelled up and prematurely old. It is asserted that etherism has been a substantial factor in the production of insanity, but though this would seem to be only what might be anticipated, the writer is not satisfied with the proof of more than a very few cases. There are cases, too, of etherists who have *apparently* suffered no injury to health.

The *treatment* of chronic etherism is much the same as that of chronic alcoholism. The complications are largely gastric, calling for the administration during a week or longer of peptonized liquid nourishment, effervescent beverages, and it may be ice. Milk, when it can be borne, is the best food till the irritated stomach has regained its digestive power. Alcohol is always contra-indicated. Isolation is often essential.

Legislation was drastically applied, on the advice of the Royal College of Physicians of Ireland in 1891, by the Government. Ether is now scheduled as a poison, under the Pharmacy Acts, so that the drug can be now sold in Ireland only by druggists under special safeguards. The result has been so successful that the sale of ether in that country is not more than one-tenth of what it had previously been, and probably the bulk of that has been for therapeutic purposes or for use in the arts.

COCAINISM.

Cocaine, the crystalline alkaloid obtained from the dried leaves of *erythroxylon coca*, first isolated by Niemann in 1846, is at once the newest and most rapidly acting, fatal, and powerfully fascinating of known narcotics. Cocaine raises the vital temperature.

Acute Cocainism.—In general the symptoms are nausea, emesis, cephalalgia, vertigo, dilatation of the pupils with disturbed or lost vision, deafness, loss of taste and smell; lividity with profuse diaphoresis, pulse rapid (130 and upward), irregular, intermittent, weak, and compressible; respirations shallow, irregular, slow, labored, and convulsive; speech and gait impaired; muscles rigid and cramped; twitching with convulsions, respiratory paralysis, with or without delirium, coma, stupor. Its well-known anæsthetic properties have secured for cocaine, especially the hydrochlorate, a wide employment in operations on the eyes and other regions of the body, and for the relief of pain in ill-health. Matthison has published a number of fatal cases.

Chronic Cocainism.—Cocaine toxication is shorter in duration as well as swifter in onset than morphine, besides being more pleasurable in the exhilarative stage and followed by fewer troublesome sequelæ. When repeated frequently there is a tendency to raving madness. The bodily emaciation, mental disturbance and decay, and moral perversion of chronic cocainism quickly supervene and rapidly increase in intensity.

Pathologically the convulsive respiratory paralysis is tetanus of the respiratory muscles, as shown by Mosso. The great acceleration of the pulse-rate is occasioned by paralysis of the vagus. The action of the poison is chiefly on the central nervous system, first stimulating, afterward paralyzing. The peripheral blood-vessels are contracted. At first after taking cocaine, as after the chewing of coca leaves in Bolivia and Peru by the natives to give them increased power of endurance in journeys, mental and bodily strength and elasticity seem to be heightened; soon, however, if the doses are repeated often and persisted in, these give way to feelings of languor, depression, and distress, with distaste for food, disturbed sleep, and hallucinations, with rapidly more serious symptoms of mental breakdown, volitional paralysis, and inhibitory palsy. Cocaine hydrochlorate may be used as a spray in the mouth, or in the nasal orifices, as has been done in cases seen by the writer, in solution varying from five to twenty per cent. But cocaine is generally self-administered hypodermically in rapidly successive injections. Though one-seventh of a grain has been recorded as having been fatal to a man aged twenty-nine, the amount of each regular dose in the case of chronic cocainists has sometimes been as large as 10 grains three times a day. Medical men have been the most frequent victims, followed a good way behind by *littérateurs*, male and female. The practice has at times originated from medical prescriptions or experimentations, but mostly in the drug being taken as an analgesic. A considerable proportion of chronic cocainists have fallen under the dominion of cocaine in their delusive hope of thereby weaning themselves from chronic alcoholism or opiumism, while other like sufferers have fallen through innocently substituting certain preparations alleged to be curative of various ailments, ignorant of there being any cocaine in the draught. The writer has never known the attempt to replace alcohol or opium by cocaine to succeed. The only permissible administration of cocaine is for the occasion only, and even then its prescription should be deliberate and definite. The *treatment* is very similar to that adopted in morphinism.

B. Substances, the Intoxicating Capacity of which is Doubtful.

GELSEMIUM.

The dried rhizome and rootlets of "yellow jasmine" (not the yellow jasmine of Britain but of the United States), *gelsemium sempervirens*, often taken for neuralgia and toothache, is a potent paralyzer and respiratory poison. In acute poisoning the leading symptoms are nausea, perspiration, contracted pupils, pain in the eyebrows quickly followed by vertigo, pain in the eyeballs and dimness of vision, ptosis, diplopia, a feeling of weakness in the lower extremities, a staggering and swaying gait, intense præcordial agony, a spasmodic sense of suffocation with convulsive fighting for breath or irregular and slow breathing, foaming at the mouth, and finally death by coma.

Treatment.—The stomach should be emptied by the tube or pump or emetic, if the case is seen at once or soon after the poison has been swallowed. If respiration fail gr. $\frac{1}{60}$ of atropine (two minims of injectio atropinæ hypodermica, B. P.) should be injected hypodermically, and be repeated if necessary in fifteen minutes or so, or belladonna (15 drops of the tincture) may be given by the mouth. If the heart fail, aromatic spirit of ammonia or spirit of chloroform or some other stimulant should be given and be followed by prolonged artificial respiration. Alternate cold and warm douches over the head and chest have been found useful. The smallest fatal dose of the liquid extract (U. S.), containing 480 grains to the ounce, has been about three drachms.

Chronic gelsemiumism has always sprung from the relief afforded from acute pain. The great ease thereby secured has led rapidly on to more frequent and larger doses.

The more common *symptoms* are restlessness, emaciation, and listlessness. The mind is troubled by indefinite fears, while there are sensory hallucinations. Vision is disturbed, and though no organic lesion has been detected, mental decay sets in prematurely.

Treatment.—The drug should be withheld, and restoratives and nerve tonics, with coffee and nourishing soups, should be administered for a time, attention being paid also to the condition of the heart.

SULPHONAL.

This substance (diethyl-sulphon-dimethyl-methane) is a valuable hypnotic apparently affecting neither temperature, pulse, nor digestion. Though the ordinary dose is from 15 to 30 grains, the writer has known a literary man of thirty-five swallow consecutively, without troublesome sequelæ, 2½ drachms. Sulphonal seems to affect the cerebellum, producing a tendency to fall backward. Only one case of chronic sulphonalism has been seen by the writer. The leading symptoms in acute sulphonal poisoning are deep drowsiness, the patient being waked with difficulty and at once relapsing into sleep; vomiting is absent, the eyelids are shut, and the pupils react slowly to light, being neither contracted nor dilated; the skin is ashy and pale; there is a small, quick pulse; muscular flaccidity (most marked in the limbs) and loss of power are noticed; the respiration is quick, shallow, at times imperceptible; the tongue is coated and there is an offensive odor of the breath and from the body; liquids are retained in the mouth and swallowed with difficulty; there is aphasia or indistinct articulation; stupor tending to paretic ptosis and facial paralysis is present. Stevenson has recorded two cases of acute poisoning, followed by recovery, after the ingestion of fifty and ninety grains respectively.

PARALDEHYDE.

This drug resembles chloral in physiological action, but has a strengthening influence on the heart instead of the depressant influence of chloral. While strengthening, it diminishes the frequency of

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cardiac action. The flow of urine is increased, but the skin is unaffected, and there is neither headache nor digestive disturbance. In cases where there is a weak heart paraldehyde may be given instead of chloral. A case of *acute poisoning* with recovery after 3½ ounces of paraldehyde was reported in the *British Medical Journal*, 1891, II., 1,254. The woman was found unconscious, her body limp as if in anæsthesia. There was a strong odor of the fluid from her breath, with facial flush and slight contraction of the pupils, which were insensible to light. The skin was warm, but there were rapid pulse and breathing. The symptoms of *chronic paraldehyde* poisoning, as seen in a coachman, aged sixty-five, were emaciation and anæmia, with slight rise of evening temperature; the heart after a time became weak and irregular, and there was palpitation, with an intermittent and soft pulse; constipation, boulimia, and flatulence were present; there was muscular weakness with tremors, especially of the tongue, facial muscles and hands; feeble and unsteady gait, with general restlessness; sensory disturbance of "strange feelings running through the body." Mentally, there were insomnia, agitated anxiety, discontent, unreasonableness, confusion and excitement, temporary loss of memory and incoherent speech, shouting and a tendency to strip off the clothes, hallucinations of sight ("seeing strange beasts"), of hearing (seeing his death in the newspapers, he having heard his wife say she wished he were dead), delusions of being poisoned, of a woman being in his bed, of people tormenting him, of the doctor trying to kill him, and of the house being on fire. The patient was married; he had been steady and cheerful though quick in taking offence, and at times suspicious and unreasonable. A brother had died from alcohol mania in an asylum. He had taken paraldehyde, under advice, for insomnia with which he had been afflicted for seven years. In the twenty-six months of his addiction he had increased the weekly quantity consumed to sixteen ounces. The patient voluntarily entered the Royal Edinburgh Asylum where he was under treatment three months, and made a good recovery. In this case it had been designed to diminish the dose gradually, but the patient declined to take any more paraldehyde on the fourth day, after having had six drachms on the first night and four drachms on the second and third nights. After an interval of five days without any hypnotic, he had sulphonal for a few days. The temperature did not seem to have been affected (Elkins in the *Quarterly Journal of Inebriety*, October, 1894). Stucky of Louisville, Ky., U. S. A. (*Ibid.*, April, 1894), reports the case of a young woman aged twenty-two, a morphinomaniac, for whom he had ordered three ounces of elixir of paraldehyde. Five months afterward he found her taking six ounces daily, she having steadily increased the dose. The heart was not substantially affected and there were no alarming symptoms. The paraldehyde was at once cut off, and the patient was confined to bed under the care of a nurse, valerian and strychnine being given.

ANTIPYRIN.

Dimethyloxychinizin is a largely used analgesic febrifuge and hæmostatic, and though its existence has not spread over many years it has already been indulged in to excess. Carppelletti (*Annual of the Universal Medical Sciences*, 1894, Vol. II., E. 23) records the case of a neurotic female, aged twenty-three, who had been addicted to antipyrin for two years, taking eight grams (two drachms) daily, who had entered an asylum for treatment. She suffered from anorexia, insomnia, tinnitus aurium, and muscular feebleness. The dose was reduced, and potassium bromide with caffeine given. Prostration and functional disturbance followed the reduction. The writer has seen great physical havoc wrought to the nervous constitution, especially of girls and women, through the frequent and increased doses of antipyrin for headaches and pains, the original cause of which should have been traced and treated, if at all possible. A modest dose of from one to five grains has at first been taken and found effectual. Resorted to too often, the medicine has been found to lose its power, with the result that the quantity has had to be steadily augmented to secure relief. In this way, four, five, and six times the initial dose has been by and by consumed in a day or several times in a day for lengthened periods. The inevitable issue is that the pains and aches are more frequent, as well as accompanied with an intenser agony than ever, the remedy having proved in the long run worse than the original trouble to relieve which it was employed. The nervous system is thus shattered, with at times the sad sequel of complete nerve prostration tending to a life of irritable physical misery. Yet, with all this chain of attendant sufferings, the writer cannot class this drug as a narcotic. Even in large doses he has never seen it impair perception, dull the senses, obscure the judgment, destroy the moral sense, and stifle conscience, as he has seen alcohol do. Medicinally prescribed by skilled and judicious physicians antipyrin is a valuable remedy, and can be given in large and repeated doses limited to the occasion only, in a hospital where the patient is under close and intelligent observation. But the injury that has already been inflicted on not a few persons necessitates a plain warning that antipyrin is too potent a remedy to be self-administered with safety.

ARSENICISM.

Many fatal cases of acute poisoning by swallowing arsenious acid (white arsenic) powder have been recorded. The poisonous symptoms sometimes appear in a few minutes, generally in from ten minutes to three-quarters of an hour, but have been known to be delayed for many hours. When absorbed from the ulcerating skin the period of incubation is much longer. The prominent symptoms are faintness, depression, nausea, and emesis, with a burning epigastric pain increased on pressure, extending over the abdominal region, with agonizing abdominal and frequently leg cramps (particularly in the calves), violent purgation, with tenesmus and bloody evacuations, the stools being stained with bile and flaky mucus. The soot mingled with ordinary commercial arsenic varies the color of the discharges, the green being dependent on bile. There is a constricted burning feeling in the mouth, fauces, and throat; the pulse is feeble, rapid and irregular, at times almost imperceptible. The skin is at first hot, but in collapse cold and clammy. The extreme gastric and abdominal tenderness causes respiratory pain. In exceptional cases the patient does not complain of pain. After great restlessness there may be coma, paralysis, muscular spasm of the limbs, or tetanic convulsions. Only some of these symptoms may be present, and there may be remissions or intermissions. If there is free and early vomiting, recovery may take place even after an excessive dose. A fatal result usually follows a dose of two to three grains or more.

The *treatment* should consist of evacuation of the stomach, followed by copious draughts of warm greasy water or salt and water to wash out the stomach. Then repeated ounce doses of dialyzed iron, or unlimited draughts of hot water with freshly prepared sesquioxide of iron, made by precipitating tincture of perchloride of iron with bicarbonate of sodium and filtering through a handkerchief; or, in default of the above, magnesia may be given in unlimited doses. Castor oil may be given freely, or olive oil alone or with an equal part of aqua calcis, mucilaginous drinks (white of egg, barley-water, or linseed tea). Hot applications and frictions should be made externally. After the acute symptoms have subsided linseed meal poultices with laudanum or other opiate may be applied to the abdomen, and, if necessary, a subcutaneous injection of $\frac{1}{4}$ to $\frac{1}{2}$ grain of morphine may be given.

Chronic arsenical poisoning may be caused by the arsenic in wall-papers and many other articles, such as artificial flowers, the symptoms being puffy eyelids, with conjunctival redness, thirst and dry mouth, with congestion of the nasal mucous membrane, anorexia, a feeling of weight and pain at the pit of the stomach, dry and dirty-looking skin, sometimes scaly or with painful patches (eczema arsenicalis), limb and joint aches, disturbed sleep, harshness and roughness of the voice, nausea and vomiting, diarrhœa, stools tinged with blood, cough, hæmoptysis, wasting, and general debility.

When taken in gradually increasing doses (*arsenic habit*) there may be either only a few or none of the symptoms just stated, and the arsenic-eater may, on the contrary, appear quite healthy, or, like the adults in Styria, of a sallow, clear, waxy complexion with a moody expression, and sometimes indigestion. In the arsenic mines of Cornwall the miners eat the mineral freely, and apparently with impunity (Jabez Hogg, Proceedings of the British Society for the Study of Inebriety, November, 1889). The quantity taken at a time by arsenic-eaters is sometimes astonishing. A Styrian peasant took 4½ grains one day, followed by 5½ grains the next day (Roscoe). Another took 7½ grains in one day (Maclagan, *Eclectic Medical Journal*, 1864, Vol. X., p. 200). Arsenic is also taken in Britain in the liquid form of Fowler's solution (liquor potassii arsenitis), which contains four grains of arsenious acid in the fluidounce. In one adult case the initial dose was only three drops three times daily, the medicine having been then taken for neuritic pains and general debility. Usually the arsenic-habit has originated in therapeutic use of the drug, for the relief and cure of such maladies as ague, rheumatism, nervous debility, and neurasthenia. In a small proportion of cases the drug has been self-administered from the first. Notwithstanding the peculiarly firm hold which arsenic in various forms takes on certain individuals when once used for any purpose, the writer has never seen or heard of any mental damage or moral perversion supervening. On the contrary, the continuous use of arsenic (the use is never periodical except therapeutically to combat periodic diseased symptoms) appears in actively intelligent people to sharpen the intellectual powers, without depraving the moral sense. While the poison is being taken regularly, unless an overdose be consumed, as a rule there is no appearance of any departure from health. The trouble comes if the drug is suddenly discontinued, death being imminent in from twelve to fourteen days. In all cases of chronic arsenic-use the only safe rule of treatment is the gradual reduction of the dose extending over from four to six or even more weeks, the period of reduction being proportional to the average daily quantity formerly taken. Plenty of light nourishing and readily assimilable food must be given, with medicinal stimulants and nerve tonics.

OTHER NON-INTOXICANT SUBSTANCES.

There are many other poisonous non-narcotic and non-anæsthetic drugs which have been known to have left such an abiding impression on the human constitution, as to have led to the continued increasing regular consumption of them. Among these have been iodine in various forms such as the liquid tincture of iodine and the solid iodine salt, potassium iodide; one case of the former, and a number of cases of the latter, have come under the writer's cognizance. But a similar impression has also been made by less actively poisonous and even innocuous substances, such as paraffin, potassium bicarbonate (which was eaten by a female as freely as many people eat sugar), castor oil, and cod-liver oil. Epsom salts (magnesium sulphate) so acted upon a Baptist minister's wife that, from half of an ordinary dose in early life, she had so persistently increased the dose that when she died, at the age of ninety, she was taking a pound and a half a day. Geophagy, or earth (dirt or clay) eating, may be referred to here. This practice prevails over considerable areas in Africa and America, and over more limited tracts of country in Japan and Northern Europe (Pavy, "Treatise on Food," 2d ed., pp. 221–223). In the West Indies white clay has been preferred to alcoholic liquors and tobacco. The origin of this extraordinary practice seems to be unknown, though the actions of many of these dirt-eaters would point to some abnormal brain depravity. This earth-addiction is said to be incurable. Nursing mothers will take pieces out of the walls of their hut or apartment to gratify the diseased craving, and even give portions to the child at their breast to keep the infant quiet. Children begin quite young, are apt to fall into a decline and die at an early age, with dropsy as a leading symptom. If middle age is reached, dysentery generally causes death. The practice induces death prematurely. Apart, however, from the deleterious effect of this earth, dirt, or clay itself on the system, the young of the *Anchylostomum duodenale*, an intestinal parasite embedded therein, may also be swallowed, with a fatal result from the loss of blood arising from the wounding of the intestinal wall by the sharp teeth of the parasite.

C. Unintoxicating Substances.

TOBACCOISM.

A long and vigorous controversy has been waged on the merits or demerits of tobacco. On the one hand "the divine weed" has been denounced as the embodiment of all that is physically mischievous, and as morally deadly from the great part which it is alleged to play in the causation of alcoholic intemperance. On the other hand it is claimed to be innocent, wholesome, pleasing, a comforting plant which adds to the happiness while subtracting nothing from the healthfulness of life. The truth lies within these extremes. Some persons cannot smoke or otherwise use tobacco without discomfort, distress, and danger. Tobacco is not an innocent substance. It contains nicotine, an alkaloid which is a poison, destroying life in small doses very quickly. This is a volatile, thin, oily-like, acrid liquid, and fatal cases of poisoning by it have been recorded. It acts as speedily as hydrocyanic acid. The symptoms of *acute* tobacco poisoning are nausea, emesis, giddiness, faintness, weakness, pallor, mental confusion, dimness of vision, feeble and small pulse, loss of power in the limbs, and general muscular relaxation. Sometimes there are cramps, purging, tremors, prostration, coldness of skin, with cold, profuse, clammy perspiration, convulsive twitchings and other movement paralyses. The pupils are at first contracted, but afterward dilated. The odor of the patient is disagreeable.

Treatment.—Empty the stomach by pump, tube, or emetic. Give frequent doses of strong tea, or thirty grains of tannic acid, by the stomach tube if needful, to render the alkaloid insoluble. Inject hypodermically gr. $\frac{1}{25}$ of strychnine nitrate or give by the mouth twenty minims of nux vomica, with aromatic spirit of ammonia or spirit of chloroform. Apply external warmth and warm rubbing. The patient should keep the recumbent position. Hot water may also be given. Acute poisoning by tobacco taken internally is rare, as it is usually only taken in poisonous doses either inadvertently or as an enema, the latter employment of tobacco being now practically obsolete. Thirty grains of tobacco or one to two drops of nicotine would most likely be a fatal dose. Death by tobacco may occur in less than half an hour, by the alkaloid in a few minutes.

Chronic tobacco poisoning is much more frequent, though rarely fatal, owing to the subjective symptoms necessitating the reduction or complete withdrawal of the drug. Through its action on the nervous system tobacco contracts the capillary vessels, causing facial paleness and cuticular coldness. There is subsultus with general muscular trembling, while the heart labors to send on the blood supply till the cardiac vascular system also succumbs to the narcotic influence. Then there is involuntary gastric spasmodic contraction, sometimes going on to tetanic convulsions. Alcohol has an opposing action, as from its being a nervous relaxant it sets free the heart, reduces muscular rigor, and counteracts the nicotian action. It is somewhat curious that there is another characteristic in which tobacco differs from alcohol. Though every kind of animal can be acted upon by alcohol, many animals, such as goats and pigeons, can live and thrive on tobacco. The symptoms of chronic tobaccoism are usually functional, such as dyspeptic nausea, bad taste in the mouth, gastrodynia, cardialgia, and disinclination for food, dryness and congestion of the throat, fauces, and mouth, tendency to faintness, with disturbed and lessened vision. Continued excess by and by may lead to aggravation of all these and other symptoms, to such a degree as to be intolerable. What constitutes "excess" is a varying quantity. Some persons cannot use tobacco in any form without acute distress. To all such individuals the smallest quantity is excessive and rapidly injurious. Abstinence is for them the only temperance. Others are able, with enjoyment and even apparent impunity, to smoke or snuff or chew tobacco, just as many alcohol-takers live, to all seeming, long lives of healthful activity. In both cases, one cannot, however, be certain that no harm is being done. The *quantity* of tobacco consumed daily is sometimes very great, not a few tobacco-users smoking or otherwise consuming tobacco during all their waking hours. In some countries, as in Paraguay, the whole population, young and old, smoke. Tobacco exerts a powerfully *depressant and disturbing influence* on the nervous system. So potent is this influence that the writer has seen abstainers, from fifty to sixty years of age, before they had their morning pipe which steadied them, trembling in every limb and with tremulous tongue as if they had been alcohol-drinkers and were laboring under an attack of delirium tremens.

The mischievous effects of tobacco on the sight are seen in tobacco amblyopia and amaurosis, and in dryness of the upper lid, "smokers' sore eye." This toxic amblyopia arises from a subacute or chronic neuritis attacking first that part of the optic nerve trunk which contains the nerve fibres going to the region of the macula lutea. If neglected the impairment of sight and of color sense from the centre of the field over the whole of the nerve fibres leads, though rarely to absolute, yet generally to practical blindness. Dowling of Cincinnati (*Medical and Surgical Reporter*, Phila., 22d Oct., 1892) found, in 3,000 workers in tobacco works, 150 with impaired vision, of whom 45 suffered from amblyopia. Very few of the workers drank. One woman of forty, who had never used but had worked in tobacco, had amblyopia. The vision of 30 more had been gradually failing. Fernandez (*Annual of the Universal Medical Sciences*, 1893, E. 17) stating that nicotine amblyopia is very rare in Cuba, though smoking is so common and so excessive, accounts for this by the manner of use, neither in pipes nor with too much chewing. In central toxic tobacco amblyopia the ophthalmoscope may reveal only slight changes, though Noyes found, in a boy of fifteen years who exhibited trembling tired muscles and disturbed sleep, of cigarette origin, the papillæ of the optic nerve abnormally reddened at first, later anæmic, ending finally in atrophy of the disc. The sound *treatment* of tobacco amblyopia involves immediate and total discontinuance of tobacco in every form, the abstinence to embrace alcoholic intoxicants if these have been used. It is often difficult to decide, except in the case of abstainers, whether the toxicity has been of tobacco or alcoholic origin, or of their conjoint action. The general health must be improved by absorbents such as potassium iodide and mercury in minute doses, with strychnine or other nerve tonics.

Chewing seems to be the worst mode of use, though Merlin of Algeria (*Gazette Médicale de l'Algérie*, 15th Aug., 1892) reaffirms that smoking causes labial cancer, a belief strongly held by Lizars of Edinburgh and others. The writer, though he does not question the occurrence of labial and other regional epitheliomata and other malignant growths, which indeed he has seen in tobacco-users, especially in smokers of cigars and pipes, has never felt at liberty to credit those neoplasms to the pathological influence of tobacco on the blood; it is possible, however, that this may have been a contributory factor, as he has been inclined to award the credit to the local irritation set up mechanically on the lips by the pipe or the cigar (he has seen no such consequences of cigarette smoking, mischievous though that practice is, though he would expect such an occurrence occasionally), or on the mouth, fauces, and throat partly by the influence of tobacco on the secretions, partly by the acrid action of the tobacco smoke on the sensitive mucous membrane. The London *Lancet* (2d Apr., 1892) states that one hundred young men had died in the United States from smoking paper-wrapped cigarettes. This fatality has been explained by some as arising from poisonous ingredients in the wrapping and not from the tobacco itself; but on such tender constitutions tobacco must act injuriously. Tobacco-vapor seems to be powerfully noxious. Chapman of Louisville (*Medical Standard*, Chicago, Jan., 1894) describes three cases of poisoning in a tobacco-stemmery, one a girl aged nine years who died in nine days.

The writer grounds his indictment against tobacco mainly on the functional disorders which it has a tendency to induce. He has frequently seen failure of vision rapidly increasing with great nervous depression, harassing dyspepsia, cardiac disturbance and weakness with palpitation and panting, rapidly disappear when the sufferer has abandoned the use of tobacco. Largely, probably mainly through its influence on the nervous system, tobacco used continuously and in excess, in many cases when so used moderately, in addition to promoting physical discomfort and distress, impairs the senses, retards growth, and lessens muscular vigor. Seaver of California (*Nat. Pop. Review*, San Diego, Jan., 1892) records the details of a comparison between 77 non-users of tobacco, 22 irregular users, and 70 habitual users, at Yale University. In weight the non-users (in 1891) increased 10.4 per cent. more than the regular users, and 6.6 per cent. more than the occasional users. In height the non-users increased 24 per cent. more than the regular users, and 14 per cent. more than the occasional users. In chest-girth the non-user had an advantage over the regular user of 26.7 per cent., and over the occasional user of 22 per cent. In lung capacity the growth was in favor of the non-user 77.5 per cent., when compared with the regular user, and 49.5 per cent. compared with the irregular user. As regards the olfactory and auditory senses, Brodnax of Louisiana, who had been a smoker of over thirty years' standing, states that after the use of $\frac{1}{30,000}$ of a grain of strychnine, odors and sounds perceptible to non-smoking friends are not discernible by him. Dowling (*ut supra*) found that among 3,000 tobacco-smokers most of the men were flabby and anæmic. Some had smoked twenty cigars per diem. The affected generally did not sleep well, had trembling hands, and their muscles were easily tired. While these and other injurious effects of tobacco are exhibited in the person of a substantial proportion of constant tobacco-users, it is only fair to state certain benefits which in others follow its use. These are concentration of thought, mental satisfaction, protection against infection, and domestic happiness. There are persons so constituted that the intellectual powers require to be arrested and concentrated before any definite intellectual effort can be even entered upon. To such persons tobacco-smoking has proved invaluable, the advantages far outweighing the disadvantages. No other substance, narcotic or anæsthetic, is yet known which would serve this purpose and do so little damage. Were tobacco not known, the idiosyncrasies of such individuals would interfere with the achievement and excellence of their work. All with whom tobacco does not disagree realize fully the pleasure and mental satisfaction afforded by smoking. No language can accurately describe the comfort enjoyed from a pipe when one is exposed to severe weather in the trenches or the power that it has to stay the stomach-crave for food when no food is to be had; and the action of tobacco under such circumstances cannot be considered harmful. The disinfecting power of tobacco is well known, but it has been demonstrated by an ingenious series of experiments. Tazzinari of Rome (*Annual of the Universal Medical Sciences*, 1892) passed tobacco smoke for from ten to thirty minutes, through the interior of hollow bells line with gelatin containing disease-germs, when the bacilli of Asiatic cholera and of pneumonia were destroyed. Though the writer has not used tobacco in any form for many years and though he is strenuously opposed to its ordinary use, he would not think of going through a yellow-fever ward, unless after a full meal, without a lighted pipe or cigar or cigarette in his mouth. Then there are many persons, cultured and uncultured, but especially the former, who after an exhausting day's work with head or hands are so worn out and irritable that everything appears wrong from the cooking of the food to the playfulness of the children, but who when they have had a smoke are "pleased with themselves and all the world beside." This abnormal psychological state should not be neglected, as it may lead to cerebral trouble, and in such cases tobacco is as beneficial to the smoker as it is conducive to the comfort and happiness of all about him.

It has often been insisted that the use of tobacco is a fertile cause of alcoholic inebriety, but in the writer's experience this has not been apparent. It is true that a very large proportion of male inebriates have been smokers of tobacco (491 of 507 men and 70 of 93 women at Fort Hamilton, Brooklyn, 136 of 152 men at the Dalrymple Home in England, and 92 per cent. of the men with 1 per cent. of the women who have been under the writer's care); but the smoking and the drinking have generally either been begun simultaneously or, with a few exceptions, the drinking has been first. In the writer's opinion there is no characteristic or necessary causal relationship between alcoholism and tobaccoism. In a great number of instances the influences of association and social custom induce to drinking and smoking, chewing, or snuffing; the one practice does not of itself, apart from association, lead to the other. Tobacco-users who feel the need of a restorative should never resort to an alcoholic "pick-me-up," which still further depresses, but to coffee which healthily stimulates and refreshes; while for the mere quenching of thirst arising from tobacco nothing is better than sips of lemon-water. Notwithstanding all that has just been urged in exculpation of tobacco as an inebriating factor, the broad fact remains that it is a potent poison, the general tendency of which is to debilitate the system, stunt growth, and deprave function, that it cannot be indulged in except at a certain risk, especially by the young, and that general abstinence from it would greatly promote the health and vigor of the population at large.

The *treatment* of chronic tobaccoism demands discontinuance of the poison. In many cases this can be accomplished at once, and this is preferable, as the continued taking of the poison keeps up the poisonous influence. In these cases the discomfort caused by the abandonment of a long-continued practice can be greatly alleviated by aromatic, still, or effervescent mixtures, coffee, warming broths and soups, or even the occasional sucking of ginger or pellitory root (*pyrethri radix*) or an acid drop, or an orange. Medicinally, tonics, such as gentian or calumba with strychnine or nux vomica in moderate doses, are of undoubted value. The bracing up of the nervous system is aided by fresh air and exercise, bathing, at first warm, afterward cold (if the latter is followed by a reactionary glow). Moderate muscular exertion is also called for. Galvanism is useful in a few cases, though rarely needed. It is as a rule more difficult to give up smoking than drinking, especially where tobacco has been used for from ten to twenty, thirty, and forty years and upward, yet generally a resolute resolve enables the tobacconist to abandon tobacco at once, the suffering on the whole being less than from gradual reduction. The legislation in a few of the United States of America forbidding the smoking by youngsters under sixteen years of age in public places, ought to be adopted universally, and vigorously enforced. This age might, too, be advantageously extended to eighteen years. The remarkable extension of tobacco-smoking among girls and women in Britain and America, especially among the cultured classes, of which there are continuously more and more manifest indications, with its retarding influence on growth and function, calls for serious consideration, though the writer has not seen so grave mischief arising from tobacco-use in females as in males. Though the writer recognizes to the full the injurious influences of tobacco on the human constitution, unless in the extremely rare instances in which tobaccoism has developed into mental unsoundness (a condition which may be induced by prolonged excessive indulgence in even innocent articles and practices by certain neurotics), he has never seen tobacco destroy moral capacity, or lead to offences against morality or to acts of criminal violence. The poisoning has effected physical injuries, but appears to leave untouched the conscience and the moral sense.

THEINISM.

This is not observed in the form of acute poisoning, so that only the *chronic* presents itself for consideration. The tannic acid found in the dried leaves of the tea plant (genus *Camellia*, Bentham and Hooker) gives rise in many persons (in some when taken in the smallest quantities, in others only when taken in excess, *i.e.*, too strong and too often) to intractable and distressing dyspeptic symptoms, generally with constipation from the astringent properties of tannin. The alkaline crystallizable body *theine* to which tea is indebted for its main potency was discovered in 1827 by Oudry and, eleven years later, stated to be identical with caffeine by Jobst and Mulder; this was found by Wood to be present in the proportion of 2.8 per cent., on an average analysis in one hundred cases (*American Medical and Surgical Bulletin*, 1st Sept., 1894). Theine affects the nervous system primarily, and the organic secondarily. The proportion of the volatile oil, to which teas owe their aroma and which causes intoxication, is small, from one-half to three-quarters per cent. The average proportion of the tannic acid is 14.2 per cent. The quantity of nutritious matter in tea is small, of albuminoid principles 3.5, and of carbohydrate elements 9 per cent. Tea is generally drunk as an infusion, but it is also, though rarely, chewed, as in the case of two girls reported a few years ago in Boston (*Mod. Med. and Bact. World*, Feb., 1893), the latter mode of use being very rare indeed. When drunk in moderate quantities by those persons with whom it does not disagree, tea is a cheering, reviving, and exhilarating restorative. Though part of the reviving influence of hot tea when the body is fatigued is no doubt due to the warmth of the liquid, the fact that cold tea is also reviving and restorative when drunk from time to time during a heavy day's shooting or walking or riding, is proof that the stimulating properties are mainly due to the tea itself. Of all the beverages known to man, next to water and milk the best of all, probably tea is the most useful as it is the most grateful and recuperative, when prepared and partaken of judiciously. It is, at the same time, the least attended with risk. Yet it can and does, in certain persons, produce symptoms characteristic of its toxic action. The digestive apparatus and the nervous system suffer most. Acid dyspepsia (sometimes an effect of the nervous disturbance, sometimes of the topical irritation of the gastric mucous membrane) with its attendant miseries of flatulence, heartburn, nausea, faintness, dizziness, anorexia, gastralgia and abdominal pain, insomnia and depression, is not an uncommon experience. The direct influence on the nervous system is, however, the most frequently exhibited. Tremors, nervous prostration, anxiety or dread, nervous irritability, digestive incapacity, sleeplessness, cephalalgia, emaciation, mental agitation and instability, constant restlessness, and persistent melancholia, sometimes with a suicidal tendency, have all been frequently noted. In one female case the enormous quantity regularly consumed induced alarming and protracted prostration, with symptoms of impending paralysis. Though harassing physical mischief has been caused by intemperance in tea, the writer has not met with any moral obliquity therefrom.

The basis of *treatment* of theinism is abstention in some cases and reduction of strength in other cases. In the latter instance particular attention should be paid to the mode of preparation and the quality. The writer generally directs infusion for two to three minutes only, of one teaspoonful of black China for each person (about a third less of Ceylon) or a blend of China and Indian, with little sugar but a generous proportion of milk, the latter imparting nourishing material to the warming, generous, quickening beverage. When the tannin elements are producing disturbances, tanocea or any other pharmaceutical preparation which will take up the tannin in the infused tea is of marked value. When the water is hard a pinch of sodium bicarbonate before infusion is of service. In other cases a "content" of milk, sugar, and hot water is the best substitute. The favorite Russian preparation of tea with lemon, *Chaĭ*, is, especially in hot weather, a pleasant and acceptable beverage.

CAFFEINISM.

Caffeine, afterward found to be identical with the *theine* of tea, was discovered in coffee in 1820 by Runge. There is less astringent matter in coffee, as well as less volatile oil than in tea. Coffee is partaken of in the form of decoction and infusion. In Arabia (whence the most highly prized coffee comes), and the East, a decoction is made of the unroasted bean, the grounds and all being swallowed; while by European peoples the bean is roasted and is then either put into boiling water and brought to the boil before straining, or boiling water is poured upon it through a strainer to prevent the grounds from being drunk. As with tea, soft water extracts more of the virtues. Coffee, like tea, is a refreshing, stimulating, enlivening, and restoring beverage without reactionary depression, is frequently useful in the depression of alcoholism as in that after mental or physical over-fatigue, though it is more heating, heavier, and more oppressive to the stomach than tea. Both beverages are usually regarded as lessening tissue waste. Coffee is an antiseptic. Luderitz (*Medical World*, Phil., Jan., 1893), in experimenting with coffee infusions of from ten to twenty per cent., found that the bacilli of anthrax were destroyed in three hours, of cholera in four, and the streptococcus of erysipelas in a day, the antiseptic capacity depending on the empyreumatic oils developed by roasting. *Chronic* caffeinism alone has been noted. Intemperance in coffee causes anorexia, nausea, a hot, acrid taste, dry mouth, persistent thirst with barked, burnt-up tongue; feverishness, tremor, palpitation, feeble circulation, acute pains chiefly neuralgic, agonizing cephalalgia, a constant fear of falling, emaciation with pinched features, with vision so blurred as to make everything appear of a hazy brownish hue. Dyspepsia and liver disturbance are conspicuous. Though these symptoms of caffeinism produce extreme discomfort and make life a burden, there is neither the paralysis nor the anæsthesia of alcoholism. *Pathologically*, the liver and the digestion afford the most prominent disorders, there being in some cases a tendency to melancholia and suicide.

The *treatment* must be heroic, coffee being at once abandoned, and light, nourishing food (especially in a liquid form as in broths and soups) being given. Frequent effervescent saline draughts are called for, with nux vomica in small doses in slightly akaline menstrua. There are many persons who cannot take coffee without great discomfort and suffering. Such individuals should entirely avoid the use of this stimulating and mentally rousing beverage, which is so valuable in the treatment of alcoholism and morphinism, as well as in the relief of paroxysmal asthma.

II. NARCOMANIA (THE DISEASE OF INEBRIETY).

Having considered the various injuries inflicted on the human constitution by the toxic influence of inebriating and non-inebriating substances which have been taken to excess, we now proceed to inquire why human beings "take to drink" and similar articles. Having described the poisoning, we go now to investigate why the poisoned continue to use the poison. The key to the solution of this problem is to be found in certain congenital abnormal defective or acquired morbid conditions, which render a minority of mankind peculiarly liable to alcoholic, opiate, or other addiction. The chief characteristic symptom of the addiction itself is the latter's inability to refrain from indulgence to excess. It was formerly believed that drunkenness was always voluntary, drunkards having been punished by scourging, the pillory, imprisonment, torture, and even death; but the failure of penal procedure to either prevent recurrence or to effect a cure has led in recent times to a reconsideration of the whole subject in the light of clinical observation and pathological science, with the result that a physical aspect of intoxication has been revealed, and that the phenomena of drunkenness have been recognized as frequently the manifestation of an abnormal mental state. The overpowering impulse, crave, or craze for excessive indulgence in inebriants is not so much for the inebriating substance, which is often utterly abhorred by the taker, as for the satisfaction, fleeting though that prove, afforded by the intoxicant. The drug may be, often is, loathsome to the taste, yet the user is powerless to abstain though he curses himself and it at every mouthful. Nor is the mania for alcohol only. In Western peoples this is the inebriant in most common social use and most ready to hand, so it is the main form of inebriate addition. But opium, morphine, chloral, cocaine, chloroform, ether, and other narcotics and anæsthetics have also, especially of late years, been largely employed for inebriating purposes. So the writer, in the absence of any other term which, while indicating the essential narcotic character of the morbid phenomena, is comprehensive enough to include every variety of intoxication and anæsthesia, has ventured to apply the term Narcomania (*ναρκή*, torpor), a mania for intoxication by any intoxicant. He has also, to meet the common confusion of the meaning of the words "drunkenness" and "intoxication" with the word "inebriety," suggested the restriction of the last-mentioned word to true narcomania (or mania for intoxication by any intoxicant). A drunken act is no more the disease of inebriety than is a violent deed the disease of insanity. Intoxication and murder may take place without any apparent trace of an underlying and causative diseased condition; but a considerable proportion of drunken offences are the product of the disease of inebriety (or narcomania), as a substantial proportion of fatal assaults are the product of the disease of insanity. Unless the word "thirst" be applied beyond physiological and pathological significance, the term dipsomania is a misnomer. Physical thirst is not experienced by many diseased inebriates, unless after a debauch; and it may exist in drinkers who are not subjects of the disease of inebriety, who are strictly limited in their potations. Inebriety may be briefly defined as a constitutional disease of the nervous system, characterized by a very strong morbid impulse to, or crave for, intoxication.

Relations of Inebriety to Insanity.

Inebriety (narcomania) is a disease of the nervous system, closely allied to insanity, with which latter malady it has many points in common. So close is the relationship that some authorities, and the jurisprudence of some countries, class habitual narcomania as a variety of insanity. In many features there is a close resemblance between the two diseases. The past history of both is alike. Not a century ago insanity was regarded but as possession by a devil, or a divine punishment of sin. Accordingly, incantations, charms, spiritual exhortation, stripes, and other painful primitive processes formed the treatment pursued with lunatics who were exposed in Bedlam to the inquisitive gaze of the curious. In Bokhara Lansdell, so late as 1888, found maniacs while chained by the ankle kept violently jumping and dancing about, to "drive the devil out of them" ("Chinese Central Asia," H. Lansdell, D.D., 1893, I., 66). So has it been with inebriety. The inebriate has been fined, imprisoned, flogged, made an object of ridicule in the "stocks," expelled from the church, and treated generally as an intentional sinner, with the same miserable failure as was formerly experienced in dealing with the insane; but the recognition of a diseased condition underlying the eccentric doings of maniacs and drunkards, while substituting humane for barbarous treatment, has cured many of the insane who were formerly incurable, and has also (though at a later date) already largely operated with as good an effect on inebriates.

The *causation* of inebriety and insanity is practically the same. Both neuroses are transmitted, and both can be acquired. Both may arise from organic disease as well as from functional disturbance. Broadly speaking, the predisposing and exciting causes are identical. There is a toxic narcomania as there is a mental alienation of alcoholic toxicity. The remarkably intimate relationship of inebriety to insanity was curiously typified in the old Sanskrit words *mad*, one of the meanings of which was "to get drunk," and *madhu*, which meant "intoxication" and also "insanity." Intoxication is insanity in brief. In the first exhilarative stage there are the excitement, elation, and grandiose exaltation often seen prior to the onset of general paralysis and insane paroxysms. In the second stage motor coördination with automatic or semi-automatic ideation and emotion, mental confusion, passionate outbreaks, violence, and indecencies of insanity. In the third paralytic stage there are the gradual paralysis of thought and speech, the fatuous dulness, depression, helplessness, and finally the "dead drunken" comatose unconsciousness, when the drunk person is kept alive, all the senses being obliterated for the time, only by the automatic action of the organs of circulation and respiration. Extend these stages over a long term of years, as in chronic alcoholism, and there is presented to view a career of mental alienation which is known as the chronic insanity of alcohol. Drunkenness was described by Aristotle as voluntary madness, though modern research has shown that it is frequently involuntary. Maudsley aptly says that the influence of alcohol on the mental functions is an abstract and brief chronicle of the history of insanity, as the action of the poison upon the nutrition of the nervous centres is quick and transient, and that the spreading of the poisonous action over years by the drunkard exhibits a chronic and enduring insanity, which, even if stopped by death, is continued in the lives of the descendants. Various phases of mania are at times manifested in the career of some "moderate" drinkers and in occasional excess. The writer has known a generally "moderate" and excellent man, sometimes when dining out make a complete exhibition of himself by foolish, frivolous, contentious words and deeds, at times having to be restrained from perpetrating a criminal act, yet who, next morning, when the toxic influence on the brain had passed off, was once again his sober, dignified, calm, circumspect, and intelligent self. In the mania of occasional excess, "mad" acts, at times with a serious issue, have characterized the kindest and calmest of men. In the mania of periodic inebriety, the usually sedate and upright man or woman may be unconsciously guilty of offences against the law, which surely indicate a temporary mental aberration. One man was transported from England for repeatedly, when intoxicated, having stolen a Bible, others have always when drunk stolen kettles, shoes, spades, horses, etc.; while one woman, a laundress, was sentenced to prison two hundred different times for stealing a tub while under the influence of alcohol.

Pyromania, by its very name indicating mental unsoundness, is developed in some individuals when affected by strong drink. The writer was often kept awake half the night by a friend who, when "in drink" used invariably to take his stand with a loaded pistol outside the writer's bedroom door and keep calling on him to open the door and be shot. The manias of confirmed inebriety are numerous: kleptomania, pyromania, etc., ending in some cases in the general insanity of narcotism. The delusions and hallucinations of delirium tremens constitute a mentally unsound condition during which temporary insanity, suicide, and murder have been involuntarily committed. In the outbreaks of alcoholic epileptic mania, in the wild paroxysms of *mania a potu*, in the narcotic persecutions-mania with its occasionally harassing phenomena of double consciousness, and in alcoholic dementia, are seen intimate relations and analogies. As analogous, too, are post-inebriate and post-alcoholic phenomena. After years of abstention a reformed drunkard may have a recurrence of drunken bouts, or he may, if he has remained a consistent abstainer, die of far-back alcoholic lesions; just as, years after his abandonment of alcohol and cure of alcoholic mania, he may have an insane attack from disturbance of mental balance by some slight excitation which would probably have left him of sound mind, but for the brain instability induced by his old excesses. As the lunatic generally believes himself to be sane and his friends to be insane, so the drunkard usually thinks he alone is sober and all around him are drunk. Periodicity is common to insanity and inebriety, so are homicidal and suicidal mania, and melancholia.

The *etiology* of both diseases is very much the same; their *pathology* is yet obscure; there are many border-land cases in which it is often difficult to determine whether there was an insane or an inebriate origin; inebriety may induce insanity and insanity inebriety; and the sound *treatment* of both maladies is based on practically identical lines, though inebriates should not be associated with lunatics, as the successful treatment of both would thereby be hindered.

Forms of Inebriety or Narcomania.

Inebriates may be divided into two great groups: (*a*) periodical; (*b*) habitual or constant.

PERIODICAL INEBRIETY.

Occasional drunkards are generally, so to speak, accidental, persons who as a rule either abstain or limit their drinking. They are mostly to be found among the alcohol-takers, more rarely among the opium, cocaine, or allied users; and become intoxicated only at a birthday or other festivity, or when exhausted by over-fatigue or by bodily disease. Of 600 inebriates treated at the Fort Hamilton Home in Brooklyn ("Statistical Report of 600 cases of Alcoholic Inebriety," Mason, New York, 1882) 242 were of the periodic type. Of 377 discharged from the Dalrymple Home, England ("Report," London, 1895), 113 males were periodical inebriates. The habitual or constant were therefore largely in the majority. In the writer's experience of over 1,500 cases, the periodic, on the other hand, were in excess by one per cent. This difference has probably chiefly arisen from patients frequently consulting the writer long before making up their mind to enter a home, the periodic in many instances developing later on into the habitual form. Among women the prevailing inebriate periodicity is that of functional periodic narcomania, mainly menstrual. Many subjects have an uncontrollable impulse to intoxication during two days or so prior to, others during, others again for from two to four days after, the menses. The nervous perturbation in these cases (most of whom have had a transmitted neurosis) during the menstrual period has been so intense as to upset temporarily the mental equilibrium and abolish control. In not a few instances, if the subject can only be kept from inebriating articles for a few days, the inebriate impulse fades away and there is no danger of intoxication till the next menstrual period approaches. During the nerve storm there is a pathological condition which craves for the calmative influence of some anæsthetic, alcohol bringing the speediest and surest relief for the time being. In this way the drink-crave is often gradually set up, from the yearning of the whole excited nervous system for the alcoholic comfort which it had once experienced. The symptoms range from simple intoxication up to indecent exposure (such as rushing out and dancing in the streets in a night-dress) and raving violence, especially against the husband, who has to take refuge to escape assault. Inebriate periodicity is not limited to females, though it is more strikingly manifested in them than in males. Nervous periodicity affects all persons more or less. There are periods of action and inaction, exhibited for example in the alternation of wakefulness and sleep; of the vital temperature which, like the atmospheric temperature, rises and falls in regular rhythmic course. So also with the brain, there being excessive discharges of nervous force at regularly recurring periods. Inebriate periodicity may be regular or irregular. The latter is markedly seen in the inebriate outbreaks excited by acute diseased conditions, and in those dependent on the mental depression or exaltation arising from atmospheric, telluric, and climatological environment. Regular inebriate periodicity, which is chiefly alcoholic, assumes mainly a chronological form. Weekly inebriate periodicity is often met with, and if the subject is kept from drink for from twelve to thirty hours he is generally free from the drink-impulse till the following week. Sometimes the inebriate periodicity is fortnightly, four-weekly, quarterly, half-yearly, or yearly. Another form is lunar periodicity. There are also periodicities of occasion (the inebriate indulging whenever he has the opportunity of doing so), one variety of which is the periodicity of pay, as seen every Saturday, Sunday, and Monday when the workman's pay-day is Saturday; another variety being the periodicity of pension, many pensioners as they receive their quarterly instalment at once "improving the opportunity" by getting drunk and repeating the performance till the money gives out. Seafaring periodicity is yet another form of inebriety exemplified in the drunkenness of some sailors (1) going on shore after a voyage; (2) on the eve of departing on a voyage; and (3), often among passengers a day or two before reaching land, particularly on returning home. In the last instance persons who have no one awaiting them, or no duty demanding immediate attention, are apt to be so nervously excited as to be liable to be strongly impelled, on landing, to do something unusual, which nerve-storm is rapidly calmed for the moment by alcoholic intoxication. Such individuals would have found a better and safer calmative in a Turkish bath.

Commercial Inebriate Periodicity.—A periodicity met with among business men is of considerable interest. It is seen usually after a busy mail-day (sometimes mistaken for chronological periodicity, because the outbreaks occur after the weekly or fortnightly dispatch of foreign mails) and after, or during, the annual or bi-annual stock-taking. In the latter case, the sufferer becomes exhausted, excitable, uneasy, and irritable about the second or third day, and unless looked after by some friend who keeps liquor from him and administers appropriate medicine and good nourishing soups and other nutritious and readily digestible foods, listlessness, languor, and melancholy gradually so depress him that he is apt to "drown his sorrows in the bowl," a strong internal undercurrent of morbid feeling during all his nervous disturbance keeping drumming in his ears that an intoxicant or opiate would yield him immediate ease. Another form is seen in the stock broker and the speculator, specially the latter, whose anxiety and suspense as to the fluctuations of the market and the success or failure of a large venture so unsettle the brain and nervous system, that he is apt to fly to an anæsthetic for an evanescent forgetfulness of his intolerable nervous agitation and fears. The nervous irritation and disturbance, with the alcoholic nerve-poisoning, in many cases induce brain softening and other disastrous results. One such case was a healthy, robust, and temperate merchant, who at thirty-five years of age began to speculate. He took to drink simply in order to steady his agitated and anxious brain, gradually having to increase the alcoholic dose to secure the desired comfort, till soon after the loss of a third fortune he was attacked by incurable paralysis antedated by from fifteen to twenty years.

Climatological periodicity has already been hinted at. Some persons, as soon as they remain for a few hours in a certain climate, experience a continuous and rapidly increasing impulse to drinking and intoxication. They have extreme difficulty in resisting this drink-impulse for a time, but generally yield to it unless they leave the place. By some persons this morbid impulsion is felt only in a dry bracing climate, by others in a moist relaxing climate. Others again are affected in this way only at the change of season, some in spring, some in summer, some in autumn, some in winter. Even the "glorious east wind" of Kingsley operates powerfully to intoxication in certain constitutions. Oppressive sultry weather is often, from the electrical state of the air, productive of extreme irritability and unrest in many diseased inebriates, a nervous, highly strung condition during which the greatest watchfulness is required to ward off a drunken bout. In genuine hospitals for the treatment of inebriety, the cares of the medical staff are greatly increased by the extra vigilance with which they are compelled to observe the varying moods of the patients in such close weather.

Premonitory Symptoms of Periodical Paroxysm.

While in a few cases the inebriate periodical paroxysm may, like some cases of epileptic explosion, suddenly and unexpectedly manifest itself, in by far the larger number of cases there are premonitory symptoms which, to the intelligent eye, denote the coming inebriate nerve storm. There is usually a longer or shorter overture of intense nervous irritability and excitement, with præcordial distress or sensation of sinking, the uneasiness and unrest being in general accompanied by lowness of spirits. In some periodical inebriates there is headache with disturbed or little sleep, sometimes also groundless suspicion, or an indefinable dread of some vague impending calamity. In many cases a prominent feature of the periodic prodromes is the spasmodic gulping down of solid and liquid food, the affected person being unable to eat and drink in his ordinary manner. All these, or some of these symptoms, go to constitute an *aura* as characteristic as is the epileptic *aura* in an arm or in an ear. If the inebriate patient can be restrained from drinking for a few days, the nervous paroxysm usually subsides till the recurrence of the period.

HABITUAL OR CONSTANT INEBRIETY.

Though it is now generally admitted that periodical drunkards are subjects of an imperious diseased impulse or influence, habitual or constant drunkenness is to a large extent believed to be merely a vicious indulgence which can be abandoned at the pleasure of the drunkard. Even if this were so at the outset, if drinking has been at the beginning ventured on for mere excitement or a new pleasurable sensation or from subservience to fashion, in many cases a chain of pathological tissue-changes has been set up which admittedly constitutes a truly diseased state of brain as well as of body structure. At first the intoxication may have been (it is not always) a voluntary act, but as the frequency and length of each such act have increased, the boundary between the physiological and the pathological has been unconsciously traversed, the once voluntary act having now become involuntary, or at least the tendency to this act of poisoning (which intoxication really is) has morbidly attained such an accession of force as to render resistance to the potent tendency increasingly difficult. The repeated and lengthened paralytic anæsthesia so affects the brain and nerve-centres that the capacity and activity of the higher centres are lessened; the perceptive, reasoning, directing and controlling powers being so benumbed as to leave the continuance of life to the automatic performance of vital function. Apart from the toxic inflammation and irritation of visceral and other organs, the gradual crystallization of occasional intoxicated acts into continuous excess combines with the pre-alcoholic neurosis or mental deficiency to present a gradual warping of intellect and down-grade of *morale*, as clearly symptomatic of pathological degradation as are the night sweats, the pleural effusion, and the anasarca of alcoholic phthisis, pleurisy, or renal disease. Besides all these cases, there is a great body of constant inebriates who suddenly, from abstemious habits and from a well-ordered mode of life lapse into steady drinking to excess, after nerve shock from accident or disappointment of some kind, or some other exciting influence. These have no preliminary interval of regular or irregular inebriate outbreaks. The drink impulse is developed as it were in a moment and, unless the subject is cured, continues its course, accelerated and intensified by the addition of the brain deterioration from the action of the poison itself on that organ, to mental dissolution and death.

SOCIAL AND SOLITARY INEBRIETY.

Inebriety may be *social* or *solitary*, whether it be periodic or habitual, some inebriates drinking only in companionship, others never when in company, only when alone. On the whole, opium, morphine, chloral, chloroform, ether, and cocaine inebriates display the solitary form, the majority of alcoholic inebriates the social form. On the mental idiosyncrasies of the individual alcoholic inebriate mainly depends the sociability or the secrecy of the indulgence.

Etiology.

All the factors concerned in the development of intemperate and drunken acts are included in a proper consideration of the etiology of inebriety. These contributory influences are, for practical purposes and for the elucidation of the genesis of narcomania, best considered in two great groups: *predisposing* and *exciting*, the former being the conditions rendering the individual person liable to be affected by the disease, the latter the influence excitant to a paroxysmal or constant impulse, crave, or craze for intoxication, which is the characteristic feature of the disease. All through, it should be borne in mind that some conditions or circumstances may be at once predisposing and exciting.

PREDISPOSING CAUSES.

The influences which may be regarded as tending to a liability to be affected by narcomania are those of sex, age, religion, race, climate, education, pecuniary circumstances and social state, occupation, the marriage relation, heredity, alcoholic and insane temperament, associated habits, other diseases, head and other injuries, diet, bad hygienic conditions, and intoxicating agents.

Sex.—In the United States, at the Fort Hamilton Home, of 600 patients, 93 were women. In England the proportion of women is much larger, probably about 1 to 3 of men; but of prisoners convicted ten times and over (mostly habitual drunkards) the women are nearly double the number of the men. These chronic drunkards are practically diseased or narcomaniacal drunkards. The functional crises peculiar to the sex, menstruation, puberty, parturition, lactation, and the menopause, exert a specially potent influence in the evolution of the disease of inebriety, to say nothing of the quicker and sharper brain activity and excitability of woman.

Age.—Of 600 patients at the Fort Hamilton Home 116 were between thirty and thirty-five and 118 between thirty-five and forty. Of 377 at the Dalrymple Home 196 were between thirty and forty, the average age on admission having been 35.82 years. In the experience of the writer and in all the records with which he is acquainted, the average age of more than one-half of the whole number has been between thirty and forty, the period of liability of from forty to forty-five coming next, followed by that from twenty-five to thirty; and this again followed by that of from forty-five and fifty. There is a sudden fall above fifty, till beyond sixty-five years there is only half of one per cent. The proportion of those under twenty years was about 1¼ per cent. In England the writer has seen cases in individuals under fifteen years of age, while even in children from five years old and upward, he has found the narcomaniac impulse or crave unmistakably present. Children of seven years and older have developed symptoms of delirium tremens. Intoxicants of all degrees of potency should be *absolutely excluded from the dietary of childhood and juvenescence.* Fatal accidents at foot-ball and other games have occurred through the boisterousness resulting from narcomaniac excess. Many young persons have inherited the inebriate diathesis and a small quantity of the weakest liquor may light up this morbid inheritance to activity. In France and Austria this youthful inebriety has attracted attention, while Garnier, medical head of the Paris police staff, points to the fact that during the past ten years the number of youths committing violent offences has greatly increased. These are mainly the product of ancestral alcoholic, mental, and moral degradation (*Quarterly Journal of Inebriety*, April, 1892). A question of considerable importance here presents itself. Is there a *climacteric* in the disease of inebriety? Joseph Parrish (Proceedings of the Society for the Study of Inebriety, Eng., No. 13) believes there is, and places it at generally between forty and fifty. The writer agrees, but locates the climacteric fifteen years later in life, between fifty-five and sixty-five as a rule. In one male case this occurred at fifty-eight, in another at sixty, and in a third at seventy. In these and other cases, no external or moral influence had anything to do with the reduction or cessation of intoxication, the desire to drink to excess simply ceasing so that the drinker became indifferent to liquor. These were all three cases of periodical inebriety with respectively: (1) an epileptic neurosis (the epileptic explosions did not yield with the loss of the crave); (2) a maternal inebriate inheritance; (3) no known morbid heredity, alcoholic or non-alcoholic. There was also another periodic case, a lady of sixty-five, whose length of addiction had been thirty years. The writer has seen this inebriate climacteric only in alcoholic cases. The climacteric appears to set in when the nervous periodicities become faint, when the neurotic susceptibility weakens, when the vital energy diminishes, and when the force of the passions abates. This seems to afford an explanation of the fact that few persons become drunkards after sixty, though the writer has known two cases above eighty. Though the drink impulse is much weaker after the inebriate climacteric, the liability to intoxication is not abolished, and inhibition is also lessened. In the bulk of cases, periodic has developed into habitual drunkenness, and the subject has died before the climacteric could be reached.

Religion.—The influence of religious beliefs has a varying effect in the genesis and manifestation of the disease. Though the records of most Homes for Inebriates in America and Britain (except purely Roman Catholic institutions) show a marked preponderance of Protestants over Roman Catholics, this is simply because the greater part of the population has been of the former persuasion. The writer's experience goes to point, however, to a proportionately less number of Roman Catholic inebriates, though inebriety has been and is still increasing rapidly among the latter. Religious excitement uncontrolled predisposes to inebriety, while (as in Buddhism and Brahminism) strict self-discipline and self-restraint contribute to lessen the inebriate impulse while strengthening inhibitory capacity. Mohammed, by his precepts and his example, fortified his followers against the diseased drink-impulse, though there is a large amount of non-alcoholic intoxication among them in opium, hashish, and allied inebriants. The Jewish community is remarkably free from inebriety, largely owing to the religious hygienic doctrines and observances of that great people.

Race.—The Anglo-Saxon race is peculiarly susceptible to alcoholic narcomania, and is quickly affected by the opium, morphine, and other forms of the disease of inebriety. Opium is much more poisonous and rapid in its effects in the case of Western peoples than in that of the denizens of Eastern countries, from the milder and more sluggish temperament of the latter. Belgium, Denmark, and Russia show probably the greatest proportional extent of inebriety. Britain is not far behind, neither are the United States. France (according to Garnier there is now twice as much alcoholic insanity in Paris as there was fifteen years ago), Sweden, Austria, Switzerland, and Germany are seriously affected by this alcoholic narcomania. The Italians and Spaniards are by far the most temperate nations on the continent of Europe. Owing to their versatile temperament, those of the negro race are readily excited to drunkenness, and indeed more speedily killed thereby, yet there is less of the disease of inebriety among them than among other races. Especially among the American Indians and other savage races is there a strong tendency to narcomania. While the negro when intoxicated is merry and boisterous, the American Indian under similar conditions is infuriated to madness. The latter is so dominated by the drink-impulse that he drinks himself to death, he cannot stop. Jews owe their absence of predisposition to excess in narcotics in great part to their striking racial control. The writer has seen only two cases, in England, of inebriety in the person of a Jew. In the long annals of the race the general freedom from inebriety has been a prominent feature.

Climate.—Climatological conditions have substantial influence in predisposing many individuals to inebriety. Fog and damp by their depressing action on the higher nerve-centres have this tendency, while on the other hand clear, dry weather, by giving tone and vigor, has an opposite effect. Sultry weather with the air overcharged with electricity throws the nervous system of not a few men and women into a state of disquiet. The east wind makes it an extremely arduous task for some inebriates to refrain from drinking to excess. Malarial environment seems to have a marked effect in predisposing to narcomania. The same paludal causes (whether telluric, atmospheric, or microbic) which operate to induce an attack of ague, operate also to induce an inebriate paroxysm, while in addition the malarial poisoning so depraves the blood as to engender a feeling of malaise, listlessness, and uneasy depression which craves the anæsthetic relief which opium and alcohol (often consumed together by the population of marshy districts in England as laudanum in stout) quickly afford. In certain formerly swampy localities in England where this mixture of alcohol and opium was frequently taken, the quantity consumed has enormously decreased since the districts have been thoroughly drained. Colder climates apparently predispose more to inebriety than hot climates. As a general rule the northern part of France, for example, is (with the exception of some seaports in the South) more drunken than the southern parts of France. The cold regions of Russia, Sweden, Belgium, and Germany furnish a greater crop of inebriates than the southerly and warm regions of Spain and Italy. The drink-impulse and the opium-crave are less easily resisted in the northern part of the United States and in Canada than in the southern United States, New Orleans and other seaports excepted. The writer has noted the differing features of American and British inebriety and drunkenness correspondent to the differing climatological conditions, the dry bracing electric atmosphere of the former contrasting with the more moist, dense, and depressing atmosphere of the latter. The greater nerve tension under the first-named, as well as the rapid exposure to temperature variation, largely accounts for the greater intensity and rapidity of American inebriety, the entire career of an inebriate in America from start to a fatal finish being, in the writer's experience, one-third of the duration of a similar career in England. Intoxication itself is more mercurial and boisterous, if more maddening, in America, more sodden and brutal in Britain.

Education.—Three-fourths of the cases at Fort Hamilton, over ninety per cent. at the Dalrymple Home, and nearly ninety per cent. of the writer's private cases, have received a liberal education (one-third had a university training). Crothers and others have had a similar experience. Among the most difficult of the writer's cases have been the highly cultured, whose extreme nervous susceptibility and keenness render them peculiarly liable to neurotic exhaustion and storm, when the muscular system is not correspondingly exercised and developed.

Pecuniary Circumstances.—All experts in the straightforward and open treatment of inebriates are at one as to poverty being generally an effect, not a cause of drunkenness, but in some cases the despair and depression produced by scanty resources have operated to induce a morbid brain-state which impels to intoxication. The writer's belief is that riches proportionally oftener induce this diseased condition, from the decline of nerve vigor arising out of insufficient motive for sustained exertion.

Occupation.—Idleness is probably the largest predisposing factor as seen in England, forming nearly ten per cent., but in the United States is much less common, owing to the less numerous leisure class. Next to no occupation come callings involving tension from exhausting brain-work, serious and anxious responsibilities, and uncertainty of demand, such as the learned professions, especially the profession of medicine. Sedentary occupation predisposes more certainly than active out-door life.

Marriage.—In men there is no great difference in the proportion of single and married, but among women the ratio of the single to the married or widowed is broadly 1 to 6.

Heredity.—In Fort Hamilton 265 in the 600 showed a family history of inebriety, and 38 of insanity; at the Dalrymple Home 202 of inebriety, and 377 of insanity; Crothers had 225 in 500; the writer 755 in 1,500. On the whole, parental inebriety or insanity (one or both) was present in two-thirds of the cases, grandparental in a twenty-fifth. Bevan Lewis found an ancestral history of insanity in twenty-seven per cent., while including epileptic and other neuroses there were thirty-seven per cent. with a neurotic inheritance. Apart from disease-inheritance generally, various alcohol-born maladies are transmissible. The writer has seen a case of inherited alcoholic gout in a female life abstainer, who at forty-two years of age had an attack of acute gout in the great toe; the patient's father had been a free drinker of port wine during the intervals between his frequent gouty seizures. So with the disease of inebriety, the inebriate neurosis or diathesis. The strong intoxication-impulse has been manifested at a very early age in some instances. Children of four, five, six, and seven have drunk eagerly and to drunkenness on the very first occasion when drink was given to them, while the other children with them have evinced no such eagerness. This morbid impulse, unless resisted, tends to grow stronger. Even when resisted successfully, this transmitted abnormal nervous deficiency may remain latent, through a life of abstention from intoxicants, ever ready to be lit up into activity on the application of an exciting provocation. Inebriate heredity may be homogeneous (similar) or heterogeneous (dissimilar). In the latter, inebriety may in the offspring be transformed into various other nerve disorders, such as epilepsy in one descendant, idiocy in a second, insanity in a third, hysteria in a fourth. The similar form may consist in either a propensity to intoxication without any apparent exciting temptation, or an intoxication impulse on being given a little liquor. Conversely, the altered transmission has descended from a non-inebriate source in the shape of inebriety. Inebriate heredity may be immediate (from one or both parents); or mediate (from one or more grandparents). It may also be single (when one) or double (when more than one) parent or grandparent has been the origin. The bulk of authorities agree as to inebriate inheritance from inebriety, or from some other form of mental degeneracy. The following is the inebriate genealogy of generations given by Morel: 1. Alcoholic male excess, depravity and brutish disposition; 2. Alcoholism, maniacal attacks, and general paralysis; 3. Sobriety, but hypochondriasis, persecutions mania, and homicidal proclivity; 4. Feeble intelligence, mania at sixteen, stupidity, idiocy, impotence with race extinction. Weismann has denied the transmission of acquired characters (somatogenic as distinguished from blastogenic), holding that the latter are handed on unaffected by the bodily environment; but the writer's observation of inebriate families directly opposes Weismann's contention. Demme has compared the health history of ten families of drunkards with that of ten temperate families. The direct progeny of drunken parentage amounted to 57, of whom 25 died from insufficient vitality, etc., in their first year, 6 were idiots, 5 dwarfed, 5 epileptics, 1 choreic and idiotic, 5 had hydrocephalus, hare-lip, and club-foot. Two of the epileptic youths had transmitted alcoholism. Only 17.5 per cent. enjoyed ordinary health in childhood. Of the non-drunken stock there were 61; only 5 died from deficient vitality, 4 suffered from curable nervous affections, and 2 had congenital defects, 81.9 per cent. were sound in mind and body during childhood and youth. Clouston (Annual Report of the Edinburgh Royal Asylum, 1889) points out that an hereditary condition has been set up in many brains, causing feelings and cravings to outrun control.

Temperament.—The nervous temperament (two per cent. passionate) has largely predisposed to narcomania.

Associated Indulgence.—The writer has not seen, unless in exceptional cases, that tobacco-use predisposes to inebriety, though the associations connected therewith may lead to drunkenness. Opium, chloral, or other narcotic addiction is usually associated with alcoholic inebriety.

Other Diseases.—Syphilis, epilepsy, chest disease, rheumatism, gout, and uterine diseases, as well as other maladies, exert an influence in inebriate predisposition. Injudicious and unwholesome *diet* (such as white bread and tea continually used) and bad hygienic conditions, by depraving the blood, are also potent predisposing factors. Intoxicating substances themselves, even when consumed "moderately," frequently similarly predispose by their toxic anæsthetic weakening of inhibition, by tissue-degradation, depravity of function, impairment of brain capacity, and volitional palsy.

EXCITING CAUSES.

Nerve shock (domestic or commercial worry, religious or other hysteria, marital disparity, good or bad fortune, disappointed affection, and fright) excites to inebriety in from six to thirty-six per cent. *Traumatism* is a substantial exciting cause, head injuries especially. Of 123 patients at Fort Hamilton who had sustained an injury to the head, 71 became habitual and 52 periodic inebriates. Of this class of injuries, in the writer's experience fracture of the skull and falls upon the head have been the chief. Of *other diseases* dysmenorrhœa and amenorrhœa occupy a prominent place; so do dyspepsia, gout and rheumatism, epilepsy, syphilis, head and meningeal affections, and insanity. *The sexual function* is responsible for much periodic excitation. At puberty and the menopause, during pregnancy and in parturition, as well as at the menstrual periods, women are specially liable to inebriate excitation. Marital excess affects both sexes. *Climate* excites to the inebriate paroxysm, causing nervous storm which is temporarily stayed by intoxication. *Idleness and overwork,* mental and physical, are equally excitant, especially in adolescence. The *monotony* of *routine* and *dulness* contributes a considerable excitation, particularly in women. The most powerful exciting influence is, however, exercised by *intoxicants*, as is markedly seen in sudden excitation to excess on the administration of intoxicating liquor to some reformed drunkards—ecclesiastically at the Protestant sacrament and medically as a therapeutic remedy.

Pathology.

There is a physical antecedent to each sensation, in health normal, in disease abnormal. Narcomania of alcoholic origin, immediate and remote, has been considered under the pathology of alcoholism. Voluntary drinkers drink from custom or fashion. Involuntary inebriates drink because they have a strong morbid impulse to, or crave for, intoxication. In the latter the physical antecedent may be: *(a)* depression, as after exhausting hemorrhage, as occurring in melancholia, or in the nervous crisis of "neurotics;" *(b)* morbid exaltation; *(c)* pathological unrest; *(d)* pathological perversion of sensory-ganglionic function causing inebriate "crave;" *(e)* pathological perversion of desire; or *(f)* pathological inebriate impulse. Functional disorder of some kind is frequently the cause of the paroxysm, sometimes preceded by structural pathological degeneration, the liver, stomach, and brain being in many cases involved. The disturbance of function is the usual pre-paroxysmal pathological antecedent. In constant inebriety, there is imitative automatic repetition of nervous discharge. The inebriate diathesis may be either inherited or acquired. The inheritance may be of either, *a*, defective inhibition, *b*, mental instability, *c*, volitional enfeeblement, *d*, special proclivity to intoxication, or *e*, an allied neurosis. The diathesis is thus from deficient brain and nerve tonicity. This diathesis, being a product of mental degeneration, may be acquired, as has been already noted, through the action of alcohol and through any other agency causing mental degeneracy, such as epilepsy and unsound brain states. In the writer's opinion, there is an inebriate centre in the higher nerve-centres, which future pathological research may disclose. He is also disposed to think that there is an intoxication-centre, and that drunkenness is something more than the result of alcoholic paralytic inefficiency, though the cortical motor areas are early affected.

Treatment.

This has been practically covered when treating of alcoholism. To the adult as to children patients, prior to an act of drunkenness, intoxicants should be forbidden, any special constitutional taint should be attacked, tonic and brain nutrients administered, adequate and appropriate muscular exercise prescribed, while all the influences of mental, moral, and religious culture should be applied. By well-directed education and training the subjects may be enabled to continue abstinent through life, handing on to their issue a sounder body and brain than they themselves were endowed with.

Medico-Legal Relations.

CRIMINAL JURISPRUDENCE.

By the law of Mitylene, Pittacus ordained a double punishment for crime committed in intoxication (Puffendorf, L. b., 8, c. 3). But Roman law was not so severe, capital punishment being remitted when incurred by soldiers who were intoxicated. Mahommedan law does not admit a plea of drunkenness (Chevers, *Man. Ind. Med. Jour.*, London, 1870, p. 806). In the United States of America more than fifty years ago New York State classed confirmed drunkards with "idiots, lunatics, and persons of unsound mind" as regards property and estate (Revised Statutes, Tit. Cap. V. of Part II., Custody of the

Estates of Idiots, Lunatics, Persons of Unsound Mind, and Drunkards). According to Clark Bell of the New York bar the better ruling of law is that if the person, at the moment of the commission of the act, was unconscious and incapable of reflection and memory from intoxication, he could not be convicted. Crime must have motive and intention, which would be absent in such a case (Buswell on Insanity, § 446, note; People *v.* Rogers, 18 N. Y., 9 Denio; Cluck *v.* State, 40 Ind., 264; Kenny *v.* People, 31 N. Y., 330). By the New York Penal Code no act is deemed less criminal from having been done in intoxication; but when any particular purpose, motive, or event is essential to crime, the intoxication can be taken into account to determine the purpose, motive, and intent. Voluntary intoxication even to frenzy has been held not to be a defence in homicide without provocation. Delirium tremens, as a disease arising from drinking due first to "voluntary" intoxication, has been accepted as a valid defence (Real *v.* People, 55 Barbour, 551; 42 N. Y., 270; Willis *v.* Com. Va., 22, etc.). A fixed and well-defined mental disease developed from drinking relieves from criminal responsibility as does insanity (Lonergan *v.* People, 6 Park 209; U. S. *v.* Drew, 5 Mason, 28; State *v.* McGonigal, 5 Harling, 510, etc.). Evidence of drunkenness is now always admissible to explain conduct and intent in homicide (50 Barb., 266; People *v.* Hammil, 2 Park, 23; People *v.* Rogers, 18 N. Y., 9). This plea is not receivable in crimes less than homicidal. This practically leaves the matter to the discretion of the judge. Since the New York State law has required deliberation as well as premeditation to constitute murder in the first degree a capital offence, it has been ruled that even "voluntary" intoxication, if enough to render deliberation impossible, reduced the crime to murder in the second degree, which implies only intent to kill (*Med. Jour. of Ineb.*, Dyett, *Med. Leg. Jour.*, Dec., 1887). In Manitoba habitual drunkards are also civilly classed as in New York State, with "lunatics and persons non compos mentis" (Consol. Stat.).

In Germany and Switzerland there is a difference in the punishment of offences done in culpable and inculpable intoxication. Though Dutch law regards drunkenness as no excuse for crime (in the military it is of itself punishable), there is a distinction between voluntary or involuntary (made drunk by example and inducements of others). In the latter case the judge may give an extraordinary (*i.e.*, a lesser) punishment. If a person drinks to acquire "courage," his punishment is greater than if he had been sober. If voluntary drunkenness be followed by total insensibility with no proof of previous bad intent, this may operate in mitigation (Instit. Law of Holland, Van der Linden, Trans. Lond., 1828, p. 215, Sec. V., 9). In Austria the person has a less punishment if he did not become intoxicated to commit the crime. Lagodin, a peasant farmer in Moravia, while drunk killed his brother. He received only eighteen months' imprisonment for this unintentional murder, and after his discharge became the head of a temperance movement in his district; and his services have since been commemorated by a public memorial (Max de Proskowetz, Proceed. Society for the Study of Inebriety, Lond., No. 29, for Aug., 1891).

France, while admitting no such plea, deprives the indicted inebriate of civil rights; neither does Sweden, while granting divorce on the ground of inebriety. By the Italian penal code, when there is a remission of the penalty on the ground of intoxication, the actual penalty is two-thirds (in time or in money) of the punishment which would have been inflicted had the prisoner been sober.

Sir Henry James, Q. C., admits that British law takes drunkenness into account: (1) when it has established positive and well-defined insanity; (2) if it produces a sudden outbreak of passion causing the commission of crime under circumstances which, in a sober person, would reduce a charge of "murder" to manslaughter; (3) in the chronic effects of delirium tremens; this plea, however, is not to be received in minor offences. But British jurisprudence has shown a remarkable concurrent evolution of judges' charges and jury verdicts going much further in the acceptance of evidence as to intoxication and inebriety affecting responsibility.

A. *Minor Offences.*—A Scotch judge, Lord Deas, a quarter of a century ago, received the plea of intoxication on a charge of theft. In 1888 (Liverpool Assizes, May), a plea of delirium tremens was received in the case of a lady charged with stealing a purse of money, etc. So in 1891 at the County of London Sessions (*Times*, March 10th, 1891), on his own recognizance for £100 and surety for £100, to come up for judgment when called on within twelve months, a clergyman aged forty, inebriate for twenty-eight years, with a history of painful disease, head injury, and sunstroke, was discharged on the understanding that he would at once enter a Home. At the same sessions, Sir Peter Edlin also postponed sentence on an inebriate lady of fifty, accused of stealing silk from a shop and with a record of three previous convictions, to afford the friends an opportunity of getting her into a Home. The recorder for London, Sir Thomas Chambers, in 1891, gave a nominal punishment of five days (liberating the prisoner at once, conditional on the accused entering a Home that afternoon) to a male inebriate charged with inciting to an unlawful offence. Two years later, the magistrate at the North London Police Court, Mr. Rose, on August 19th, 1893, discharged an inebriate medical practitioner (to come up again if called upon), on condition that he entered a retreat for inebriates for twelve months (Scot. Reform, Aug. 26th, 1893).

B. *Major Offenses.*—In 1865 (Reg. *v.* Burns, Liverp. Sum. Ass.) Baron Bramwell, in the case of a man tried for murdering his wife, while laboring under delirium tremens and the delusion that she was in league with men hidden in the walls, charged that if through drink the prisoner's mind had been substantially impaired, a ground of acquittal might fairly arise. The man was acquitted. So was a man tried for feloniously wounding his sister-in-law while in delirium tremens (Reg. *v.* Davis, Newcastle, April 27th, 1881). Justice Stephen charged that, though drunken rage would not absolve, a secondary disease would, and any disease which so disturbs the mind that you cannot think calmly and rationally of all the different reasons to which we refer in considering the rightness or wrongness of an action, any disease which so disturbs the mind that you cannot perform that duty with some moderate degree of calmness and reason may be fairly said to prevent a man from knowing that what he did was wrong. Justice Day (Reg. *v.* Baines, Leicester Ass., Jan., 1886) laid down that if a man was so intoxicated that he did not know the nature of his act, he was insane in the eye of the law, and that it was immaterial whether the mental derangement then resulting was permanent or temporary. In Reg. *v.* Mary R. (Galway Sum. Ass., 1887) Chief Baron Palles ruled that a person, if from long watching, want of sleep, or depravation of blood was reduced to such a state that he became intoxicated by a smaller quantity of "stimulants" than when in health, neither law nor common sense could hold him responsible, as his acts were not voluntary but produced by disease. The nurse, who had murdered a patient while under a delusion in intoxication, was acquitted as insane. Baron Pollock (Reg. *v.* Mountain, Leeds Ass., 1888) held that if insane predisposition caused intoxication by a less quantity of intoxicating liquor than would make an ordinary man drunk (though the drinking of a small quantity of alcohol was a contributory cause), there was no responsibility. The accused, on a capital charge, was acquitted as of unsound mind. In an indictment for the culpable homicide of her infant by a mother who had been drunk every day and had delirium tremens (Reg. *v.* Short, *Glasg. Daily Herald*, Aug., 1889), Lord Young would not allow the case to go to the jury and discharged the prisoner, as delirium tremens was insanity and there was no criminal intention. According to Justice Wright (Reg. *v.* Littlewood, Leeds Ass., Dec., 1892) the post-paroxysmal mania of periodic inebriety absolves, and this ruling was followed by a verdict of acquittal as insane. Justice Collins (Reg. *v.* Pugh, *Staff. Chron.*, July 30th, 1892) said that drunkenness might negative criminal intent, and the prisoner, who was accused of conspiring to commit suicide with a woman who was found drowned, was therefore acquitted. In 1893 (Reg. *v.* Gemmell, Glasg. High Court, July, 1893) a prisoner on a charge of murder was acquitted as having been insane, though there was no delirium tremens but simply acute alcoholic insanity, his mental soundness having been regained after a week's abstinence in prison. The writer sees, in this remarkable evolution of a growing recognition by judges and juries of the diseased mental state of many drunkards, the approaching of the time when all intoxication (unless voluntarily induced for the purpose of anæsthetic "nerve" to commit a crime), whether temporary or permanent, will absolve, and when a like validity will be accorded to a plea of inebriety (the disease of narcomania) apart from intoxication at the moment of the committal of the crime.

CIVIL LAW.

The evidence of an intoxicated person is not received, but his evidence can be taken on return to sobriety. The writer holds further that, even in temperate intervals, the testimony of some diseased inebriates is unreliable. His evidence to this effect has been received and acted upon to the benefit of the accused in several cases. In England *confession* when the person was intoxicated, if no inducement was held out, is valid; in Scotland invalid (Best, "Prin. Law of Evid.," 7th ed., Lond., 1883, p. 479, § 529; Dickson, "Treatise on the Law of Evidence in Scotland," 1887, I., 226, § 316).

Testamentary Capacity.—In Scotland wills made during intoxication have been reduced, generally on proof of fraud or circumvention; but on the ground of extreme facility from intoxication-addiction, there have been two reductions in 1752 and 1828 (Erskine, Institutes, v. I., b. iii., t. i. and note § 16, p. 584, Ed. 1838). Justice Butt in England, in 1891 (Morgan and another *v.* Kitchen, Prob. and Div. High Court, Lond., Feb. 3d, 1891) pronounced against a later will of 1887 and in favor of a former will of 1886, on the ground that the testator had (though not intoxicated when he made the second disposition) become after the earlier date mentally incapacitated after frequent (not intoxication but) "taking his drops," and after delirium tremens. *Contracts* executed during intoxication are voidable (Stephen, Comm., v. II., p. 70, 11th ed.). Intoxication and incapacity had been held to be necessarily complete till the case of Tyler *v.* Maxwell (Court of Session, Edinburgh, 1st Nov., 1892, *vide Scotsman* of the following day), when Lord Wellwood ruled that the defensive plea of intoxication having to be total, though true in a sense, did not mean total disablement by drink. This was an action for the recovery of £5,000, the amount of a check drawn when the drawer was so intoxicated and stupid that he had to be put to bed. The action failed. *Attestation* is invalid if done by a drunk attestor, though as in other acts the presumption is in favor of validity. In a trial reported in the London *Times* of August 1st, 1892, in a case of an inebriate father appealing against a judgment of the Canadian courts which took away his paternal rights over a son under twelve, the judicial committee of the privy council decided that habitual drunkenness invalidated the *patria potestas*.

Relation of Inebriety to Insurance.

All companies reject the lives of inebriates, but the practice varies as regards ex-drunkards. Some offices refuse all such applications, others receive them on increased premiums. Especially with ex-alcoholics, permanent lesions shorten life, but with ex-opiumists there is less and not so permanent organic injury. The writer estimates that there are at least 600,000 reformed drunkards in the world, and thinks they are generally insurable lives, provided there is a minimum probationary period of entire abstention. This should be two years for every year of alcoholic drunkenness if the addiction has not been for more than two years, and one and one-half years if over two years. Ex-opiumists might have one and one-half years of probation for one year's addiction, three for two years, and thereafter six months for every two years of indulgence. All applicants should be under forty-five. There is considerable difference of opinion as to the healthfulness or otherwise of opium-taking; but the writer can come to no other conclusion than that the evidence and the facts are on the whole against opium. Chloral and chlorodyne should be ranked with opium, but chloroform and ether with alcohol. There should also be loading of the premium. This might be, to ex-alcoholics after not more than five years' addiction, an additional premium for three years older, with half a year's extra premium for each additional year of indulgence; and to ex-opiomaniacs, after two years of abstention, the premium might be payable if assuring at two years greater age, with a loading of six months for every two years more of addiction. Other individual and inherited idiosyncrasies would have to be taken into account. Mattison's proposal of forfeiture (with of course surrender value) on relapse is worth consideration. Crothers reports great loss to insurance offices from the insurance of lives, bad through narcomania ("Relations of Life Insurance to Inebriety," *Journal of the American Medical Association*). The writer has estimated that in the United Kingdom the premature yearly average of alcoholic mortality comprises 40,000 deaths directly from personal intemperance, and 80,000 indirectly from the intemperance of other persons.

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SHOCK AND COLLAPSE.

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SHOCK AND COLLAPSE.

SHOCK, as used in a medical or surgical sense, expresses a condition of sudden and more or less profound depression of the vital powers, the result of bodily injury. It may vary in degree from the slightest mental perturbation to an extreme prostration that may end in death. For convenience of clinical distinction shock is usually described under one of two forms, the first, the ordinary one, exhibiting the phenomena of torpidity, and the other those of excitement. The former is styled shock proper or torpid shock, the latter shock with excitement or erethistic shock.

Symptoms.

The symptoms of shock proper, or torpid shock, are striking and characteristic. In a typical case the clinical picture presented is one in which all the nervous energies are benumbed, and all the vital forces wellnigh exhausted. The patient appears to be stunned into an insensibility to all ordinary external impressions. Exhaustion is stamped on every feature and is manifest in every function. The expression is anxious without animation, the face pinched, longitudinally furrowed, and of ashen paleness; the lips are drawn and have a striking pallor, the nails are blue, and the skin of the general surface, but particularly that of the face and hands, is covered with a cold beaded sweat. The eyes have a meaningless, glassy, staring expression, are partially upturned under the drooping upper lids, the pupils are generally dilated and sluggish, and the sockets are darkened and sunken. Vomiting, although variable as to time, is quite constant in occurrence. Deglutition is difficult or temporarily abolished. The pulse is weak, frequent, irregular, and thready, and in severe cases can scarcely be counted. The temperature is lowered below its normal standard and registers even in the rectum 96–98° F. Erichsen found it several times below 95° F. in the mouth and axilla, and remarks that in fatal cases there may be a fall of as much as six degrees. Wagstaffe cites one case of cut throat which eventually recovered, in which the temperature was 91.2° F. The latter was doubtless, as he intimates, due more directly to the hemorrhage than to shock. The respirations are slow, shallow, sighing, and feeble, the nostrils dilated and quivering, the voice weak, low, and hoarse, questions being answered after much hesitation and with evident effort to overcome a passive, resigned, and almost helpless apathy. With all this the mental consciousness may be unclouded. Little if any pain is complained of, and in grave cases all subjective sensations are abolished. The muscular system is relaxed and voluntary movements are seldom made. The sphincters may be temporarily paralyzed, or occasionally there may be retention of urine.

These general symptoms are modified in the other well-recognized type of shock, which is termed by Travers "prostration (or shock) with excitement." Instead of lying quietly upon his back and manifesting an indifference to his surroundings, the patient tosses about in a purposeless manner, rolls from side to side, twists his body as if in extreme agony, shouts for help as if in frantic despair, and complains of fearful oppression and impending suffocation. The consciousness is clear, but is entirely preoccupied in frantic efforts to escape the impending doom. The face may even be flushed instead of being pale, and the eyes may have a peculiar wild, focusless, and "far off" look. The pulse and respiration show no marked differences as compared with the ordinary or "torpid" form of shock. There is a burning thirst present, and all fluids offered are swallowed with panting eagerness, only to be almost immediately vomited.

The jactitation gives way in its turn to tremors and tonic spasms of the muscles of the arms, legs, and body, and to hiccough. In the snatches of exhausted sleep there is restless twitching, with incoherency until active delirium, interrupted comatose apathy, clammy perspiration, convulsion, exhaustion, and death supervene. During all this ordeal the sufferer does not complain of pain, although he gives evidence of being utterly prostrated by an "indescribable sense of bodily anguish."

Although these two forms present very marked differences, they in all probability are due to the same general cause, operating under different and modified conditions, depending upon the degree and nature of the injury inflicted. In fact under certain circumstances of habit, temperament, and vitality the symptoms of one state may merge into those of the other, being more or less modified by different resisting or retaliatory influences in the individual. Generally, however, the shock of excitement occurs in cases of severe and extensive burns, of exhausting hemorrhages, of extensive crushing injuries, and in persons who have been addicted to intemperate habits.

The phenomena of absolute exhaustion so strikingly manifest in this fortunately rare class of cases has been considered by Travers to be due to a state of hyper-excitability. Horsley subscribes to this view and refers to the condition as one of "atonic hyper-excitability of the nerve centres." He, however, wishes it to be remembered "that there is almost invariably an open wound, and always some extravasation of blood, so that the possibility of the symptoms being due to pyogenic absorption never being absent, the question of its true pathology must be left entirely open."

Irregular and Modified Forms of Shock.—The other varieties of shock described by different writers can either be classed clinically under one or other of the above forms, or are examples of conditions with which pure shock in itself has nothing to do. A cardinal point to bear in mind in estimating such distinctions is the progressive tendency of reaction when the latter is once fairly started. The general rule is to have one reaction for a given injury, the ultimate result being either recovery on the one hand or death on the other.

If the original cause of shock is persistent, reaction may be irregular, imperfect, or interrupted, but we have no right to conclude that shock as such can repeat itself when once the period of ordinary reaction has passed, unless there are extra or independent reasons for the same, in the shape of fresh injuries and the like. When the patient has once recovered from shock he has, so to speak, squared his account with the particular injury causing it. If after an interval of a day or two or more of apparently established convalescence he is again prostrated, his condition is not due to *secondary shock* from the traumatism, but to the impression of some additional or consequent pathological process. Thus the symptoms that were formerly supposed to belong to secondary shock are now associated either with the well-recognized phenomena of fat embolism, or those of septic infection, or both. In both instances the invasion is as sudden and effective as that of pure shock, but in each the lines of causative distinction are, as we shall see further on, sufficiently manifest.

The *insidious* form of shock as described by Gross, in which the patient manifests a hopefulness out of all proportion to the gravity of the injury, and is, subjectively speaking, in a comparatively comfortable condition for hours after the accident, is to all intents and purposes an instance of that complete exhaustion of the spinal reflexes which cause them to be absolutely irresponsive to any irritation, while the cerebral centres are unaffected. As might be reasonably inferred, such cases usually terminate fatally with little or no attempt at reaction.

The local shock described by Pirogoff, Gussenbauer, Groeningen, and others is simply an example of restricted peripheral reflex paralysis, and imitates on a smaller scale the symptoms that belong to the more general condition.

Distinction Between Shock and Collapse.

Collapse and shock are so often employed by many authors as synonymous terms that it would appear almost like an innovation to suggest any change in their application to separate and distinct conditions. In current literature, however, there is a disposition to make them more nearly correspond with a difference of meaning which their respective definitions would imply. Thus writers nowadays speak of collapse as an overwhelming prostration that brings the patient either to the verge of dissolution or directly to death itself. Shock may be the precursor of collapse, but the latter is always an extreme degree of the former and the usual forerunner of dissolution. It would then be eminently in keeping with the respective meaning of the terms to say that a patient passed through the different grades of shock to the extreme and often final one of collapse. It would also appear to be more reasonable and convenient, and more in accordance with general usage, to associate shock with ordinary traumatism, and collapse with the more or less sudden prostration occurring in cases not strictly surgical, or rather those that come more directly under the care of the physician. Good instances of the latter would be severe internal hemorrhages from aneurisms, from rupture in ectopic pregnancy, and the like, perforation of the intestine during typhoid fever, perforation of the stomach by gastric ulcer, poisoning by irritating or paralyzing drugs, and the internal discharge of abdominal or pelvic abscesses. Collapse also conveys the idea of extreme weakness combined with prostration, and is particularly applicable to those cases that present the mixed conditions of shock from injury and syncope from the hemorrhage.

Causes.

In estimating the degrees of shock it is necessary to take into account the character of the causative influences upon which they depend. The more severe and extensive the injury and the nearer it is to a nerve centre the greater the prostration and the longer its continuance. The same may be said when any important viscus is implicated, and when a large joint is crushed, or extensive lacerations of soft parts occur, involving large nerve trunks. Extensive superficial burns by fire or steam are also attended with equally grave symptoms. It is a well-recognized fact in surgical experience that the nearer an amputation approaches the trunk the greater the danger. This is particularly the case in amputations or injuries of the lower extremities. The same rule applies in a still greater degree when the trunk itself is severely injured.

Different regions and organs of the body are peculiarly resentful of traumatism. Blows on the epigastrium have resulted in instant death, while a greater or less degree of shock is common in injuries of the breast. The slightest contusion of the testicle produces nausea and syncope, while crushing of the organ, as stated by Fischer, has been followed by severe shock lasting for hours and finally terminating in death. Blows on the neck are also followed by sudden and alarming prostration. In some respects the effects are so similar to those produced by concussion of the brain that a similar name is given to the phenomena induced, and the symptoms are generally described as associated with "concussion of the larynx." Generally added to this form is a greater or less degree of insensibility, possibly due to the indirect transmission of force to the brain and constituting the "knock-out" blow of the pugilist. Fischer believes that a paralysis of the glottis is thereby produced through a powerful impression of the sympathetic plexus, inducing cerebral anæmia. Injuries of the chest not involving the heart or larger vessels are not followed by as much shock as similar ones inflicted upon the abdominal regions. This is particularly the case in gunshot wounds.

The influences of division of certain tissues during surgical operations are quite marked in the production of shock. The cutting of large nerves during an amputation is attended with lowering of the force of the pulse beat and depression in animal temperature. Erichsen alludes to the same sinking of the pulse during section of the spermatic cord in castration. Pirogoff, before the days of anæsthesia, saw two men die on the operating-table from shock the instant the thigh-bone was sawn through for amputation. Jordan noticed similarly striking effects on the pulse and a lowering of temperature of ½° F. in section of bones. In commenting on these phenomena, so curiously associated with bone injury, Horsley concludes that they are not very comprehensible except on the assumption that it is the mode of injury which wholly determines the effect, inasmuch as the bone itself is not sensitive unless the section be carried through it near the point of entrance into the marrow of the nerve trunk supplying it. In operations for necrosis, however, shock is of no moment. Possibly the previous chronic condition of the disease in the bone develops a tolerance similar to that in other tissues of the body.

It has also been frequently noticed that all operations upon the genito-urinary apparatus, and upon the perineal and anal regions are liable to produce shock apparently out of all proportion to the magnitude or extent of the wound. The mere passage of a catheter for urinary exploration may be sufficient to cause syncope. The reasons for this apparent susceptibility are the violent stimulation of sensory peripheral nerves supplying the parts involved in the operation or injury, and their direct connection with the lumbar enlargement of the spinal cord. A similar explanation applies to all other hypersensitive parts of the body. The involvement of the trigeminus nerve in the operation of extirpation of the upper jaw is attended by very severe shock, and not infrequently the rasping of the deeper parts of the wound and the consequent laceration of the numerous sensitive nerve filaments in over-zealous efforts to remove all suspicion of malignant disease, turn the scale against the patient. "It is recognized," says Cheever, "that long-continued and large dissections on the front and sides of the neck are especially fatal," and for the same reasons.

In the exposure of abdominal or thoracic viscera the possibility of inducing severe shock is always before the mind of the surgeon. Hence operations on such parts are performed as expeditiously as is consistent with thoroughness.

The longer the operation the greater the danger of prostration in consequence. It is much better to divide a grave operation into different stages than for the sake of convenience or time-saving to do everything at one sitting. The advantages of such a line of procedure are numerous enough to establish the principle. In fact, by following out such a judicious division of labor and conservatively interrupted extension of time, operations which were formerly attended with high rates of mortality have become comparatively successful and reasonably safe. Directly and dangerously antagonistic to such views are such as have lately been advocated by surgeons who apparently pride themselves upon the number of different operations they can perform in a single séance upon one patient. Such procedures are wrong in principle and reprehensible in practice.

The modern methods of operating, as compared with those before the days of anæsthesia and asepsis, have an undoubted tendency in inviting shock. "The old method," says Cheever, "was a matter of minutes; now it is one of hours." Not only the tedious and elaborate operations of the present day are made possible, but the complicated suturing, prolonged exposure of wounds, chilling of the patient, and multitudinous dressings consume so much time that what we may gain in accuracy in one way we lose in safety in another. Too often during a lengthy and dangerous operation recesses are taken to revive the patient, and combat the successive shocks due to unnecessary fussiness in technique, in order that the patient may accommodatingly escape death upon the table.

The question of operating or not during shock is one deserving of consideration in this connection. On general principles the rule is to wait until the immediate effects of the shock have disappeared, otherwise we add to the existing prostration by the infliction of another blow. Guthrie's advice to postpone operation until the sensation of pain returned was in the direction of safety. But, better still, it is always desirable when possible to be sure that reaction is fairly established. If on account of extensive lacerations of the soft parts involving important sensitive nerves it is impossible to secure reaction, or in other words the presence of the severe irritation is itself a continued contribution to the shock, a choice must be made between the two evils by taking the lesser one of immediate operation. Under such circumstance reaction is oftentimes speedily obtained. In other cases, such as hemorrhage, strangulated hernia, and impending suffocation, in which the shock is naturally progressive, every possible risk must be taken in the direction of relief by immediate operative procedure.

The influence of anæsthesia in intensifying shock or in directly inducing it can be properly considered in its relations to this part of our subject. When an operation during shock is rendered necessary, in accordance with circumstances previously noted, anæsthetics must be administered with great care. Especially does this injunction apply to chloroform. Although many experienced operators prefer chloroform on account of the readiness with which it produces its effects, and the small quantity required, the weight of evidence is decidedly in favor of ether as the safer agent. The primary effect of the latter being stimulating to the heart is a strong argument in its favor, although, as Mansell-Moullin justly remarks, in the ordinary methods of its administration the supply of oxygen to the patient is too much interfered with. This objection can be overcome, however, by administering the ether with a reasonably plentiful supply of fresh air, at least until primary anæsthesia is induced. Oftentimes the latter will be all that will be required to complete the necessary operation.

When we recollect that pain is a potent cause of shock and may in some instances prove mortal in itself, the necessity of anæsthesia in these cases cannot be questioned. Its ability to abolish pain establishes in so far its value in controlling shock. Further than this also, when on account of the exhaustion of the nerve-centres, pain is no longer perceptible, anæsthesia may be of great benefit, for a time at least, in protecting the patient from the effects of the increased peripheral irritation, of a surgical operation of emergency.

Warren says on this point that the reason why anæsthesia has served to lessen shock after operations is probably that the nerve centres are thus protected to a certain extent from powerful irritations from without rather than the mere absence of pain.

There are some writers, Le Gros among the rest, who believe that the shock of pain is overestimated, as almost continued pain is compatible with protracted life. But there is a great difference between the gradually developed tolerance of pain and the shock of its sudden infliction. The two cases are by no means parallel for comparison of results.

Whatever may be said in favor of short operations in their relations with shock may also apply to short anæsthesia. It may be safely said that prolonged anæsthesia always contributes to shock. Even sulphuric ether, which at the beginning of an operation is a marked stimulant, soon becomes an equally pronounced depressant. Much importance is attached to the primary effect of etherization. In cases of shock in which it is doubtful whether or not the patient has reacted sufficiently to bear the operation, Dr. Nancrede is in the habit of employing what he calls the "ether test." "Everything being prepared for the operation, a little ether is administered. If this is followed by improvement of the respiration and pulse, the patient will invariably stand the shock of the operation, but if the respirations rapidly increase and the pulse runs up like a flash, I remove the ether; these patients never react."

In referring to the relatively depressing effects of ether and chloroform in inducing shock or in contributing to it, Dr. B. A. Watson mentions the curious and significant fact that after the use of either anæsthetic for more than an hour or an hour and a half the bodily temperature begins to fall and continues to do so quite rapidly. This is, according to his observation, more marked with chloroform than with ether. Another difference is that after the discontinuance of anæsthesia the temperature after ether becomes normal within an hour or so, while after chloroform sometimes twenty-four hours are required. Bearing on the same point, Savory states that in cases of forced operations during shock, he has more than once watched the feeble and irregular pulse altogether disappear as chloroform was inhaled and revive again when it was discontinued.

Railroad accidents in their influence upon shock form almost a class by themselves. There are many factors to take into account in estimating the effect of such injuries. They are inflicted with great force and suddenness; the victim is violently wrenched and oftentimes projected in the air, adding the effects of a fall to the original accident; he is not infrequently burned, scalded, or buried in the wreck, exposed to inclemencies of the weather, and suffers from a mental prostration which is in accord with the horror of the surroundings. Under such circumstances the shock is proportionately profound and reaction is usually slow. In such cases, where no apparent injury is inflicted and when the patient is merely jarred or contused, there appears days afterward a series of symptoms due, as Mansell-Moullin says, to fright combined with extensive sprains and contusions, which are insidious in their appearance and ominous in their significance. He thus describes these cases:

"After the accident the patient who has sustained no apparent bodily hurt (and this seems to be essential), appears perfectly calm and unaffected—often unnaturally so—congratulating himself on his escape; his color is good; pulse quiet; respiration tranquil; there seems nothing wrong. But at night there comes an inability to sleep, and a tendency to become feverish; the pulse becomes quicker and softer; the eye bright and restless; the extremities cool; and even within the space of three or four days, persistent vomiting and exhaustion, running on to coma, may supervene. More frequently the time occupied is much longer, and there follows a condition which has been variously called, for want of a better name, hysteria or hypochondriasis."

These cases might be classed with those usually designated as concussion of the spine. They appear, in certain respects at least, to resemble cerebral concussion in the mixed conditions of shock and direct injury to the nervous centre. The more or less permanent disability which oftentimes results gives a very plausible complexion to such a probability. By some observers it is supposed that there is some permanent alteration in one of more of the spinal centres which results in confirmed neurasthenia. Erichsen was inclined to believe that this condition, designated as "railway spine," was due to spinal anæmia. Other observers believe the lesions to be mostly inflammatory in character, the results of a slowly progressing cerebro-spinal meningitis. In the former view we must rely upon clinical evidence only, while in the latter we have actual demonstration of autopsical changes. Peter Hood believes that railroad injuries have a special influence in developing subsequent cardiac weakness.

Penetrating wounds of the abdominal viscera, with or without escape of irritating fluid into the peritoneal cavity and all cases of intestinal strangulation are, as a rule, followed by severe shock. The intestines are particularly obnoxious to injury and promptly resent it in a special train of symptoms associated with those of shock proper. Good reasons for this sensitiveness are found in the rich supply of nerves from the sympathetic system and the numerous important nervous ganglia of the abdomen. From the nature and surrounding conditions of the accidents producing direct injury or causing strangulation, the symptoms of shock are necessarily progressive, and death from shock alone may follow unless speedy operative relief is afforded. In cases of septic effusion into the peritoneal cavity there is added the shock of septic infection, which often shows itself within a few hours after the cause has manifested itself.

On the other hand it is sometimes amazing to know how much injury can be inflicted upon the abdominal contents with a minimum amount of attendant shock. The following case occurring in the practice of Dr. John H. Ripley, of New York, may serve as an apt illustration in this connection:

A policeman while arresting a criminal was stabbed in the abdomen by the latter. The wound inflicted was a few inches to the right of the umbilicus, eight inches long, and afforded a ready exit for nearly all the small intestine. The patient fell and the intestine found its way, underneath his buttoned coat front, to the loose dirt in the street. He was then transferred to the police station where he was seen in consultation by Dr. Ripley. The patient suffered so little from shock that during the cleaning and replacing of his bowel, which occupied nearly an hour, he constantly offered suggestions, and was with difficulty restrained from being the principal assistant in the operation. When the wound was properly closed the patient was sent to a neighboring hospital and recovered without a bad symptom.

Instances like the above, although not very uncommon, only serve to prove the different susceptibility to shock in different individuals.

Hemorrhage in connection with shock always adds to its gravity. Even if slight in amount it may in cases of severe prostration tend to turn the odds against ultimate recovery. The same may be said of a slight shock with severe hemorrhage. The sudden loss of even a small amount is more dangerous than a much larger amount lost gradually. The sudden spurt from a large artery has a direct inhibiting influence upon the heart itself. It is such a sudden disturbance of circulatory equilibrium that in effect it is equivalent to a direct blow. Thus the careful operator considers it his first duty to avoid the production of sudden and undue hemorrhage and promptly to arrest such as is inevitable. Especially are these precautions necessary in very young children, in old persons, and in all others who have been severely shocked by the accident necessitating the operation or have been previously debilitated by wasting diseases.

In this way we can possibly explain the difference between death from pure shock as a direct result of hemorrhage and the fatal issue from syncope due to anæmia by a more gradual bleeding.

The very young and very old bear shock badly, although the former quickly rally. Aside from tardy reaction in the aged, there are generally present some degenerative changes which add correspondingly to the gravity of the condition.

As regards the occurrence of shock in children Horsley states: "It is to be observed that, like adults, they suffer extremely from the causes of shock when those causes are applied over a large area of the body, as for example in cases of burns and exposure to severe cold. In fact their liability to suffer under these circumstances would appear to be greater than in the adult. As far, however, as operative measures alone go, there is no doubt whatever that the nervous system of the child is less influenced by traumatism than the adult. Elaborate clinical evidence in illustration of this point is not to hand, but its general truth, being supported by abundant experimental evidence, cannot be doubted, and is therefore to be considered as forming safe ground for action under circumstances of doubt."

Women are less susceptible to shock than men, are generally more hopeful, and bear pain with more resignation and less apparent suffering. Much of this disposition may be explained by a certain habit of pain to which she becomes more or less accustomed, and to the discipline of discomfort during her procreative period.

Lightning and electrical strokes generally are followed by severe shock, and recovery from the latter is prolonged accordingly. When instantaneous death is produced by these means it is probably due to a direct and overwhelming exhaustion of heart power, eventuating in cardiac spasm. The latter is part of a general rigidity of the entire muscular system, voluntary and involuntary. This condition was particularly noticed by the writer in the case of Kemmler, the first criminal executed by electricity. The instant the current entered his body he was stunned, and stiffened into an extreme condition of general tonic spasm followed by general relaxation. A second shock which, on account of imperfection in the apparatus, was rendered necessary, was attended with a repetition of the phenomena, and a happy relief in instant death. During the interval he was in a state of profound collapse. Since the perfection of this horrid machinery of death the first shock is said to kill instantly by the same means.

The influence of mental emotion in causing shock cannot be questioned. Savory says: "There can be no doubt in some cases a mental emotion alone may destroy life." It must be confessed, however, that such examples are exceedingly rare. The possibility of their occurrence is based largely on the following case related by Lauder Brunton:

> "Many years ago the janitor of a college had rendered himself obnoxious to the students, and they determined to punish him. Accordingly they prepared a block and an axe which they conveyed to a lonely place, and, having dressed themselves in black, some of them prepared to act as judges, and sent others of their company to bring him before them. He affected at first to treat the whole thing as a joke but was solemnly assured by the students that they meant it in real earnest. He was told to prepare for immediate death. The trembling janitor looked all around in the vain hope of seeing some indication that nothing was really meant, but stern looks met him everywhere. He was blindfolded and made to kneel before the block; the executioner's axe was raised, but instead of the sharp edge a wet towel was brought smartly down on the back of the culprit's neck. The bandage was now removed from his eyes, but to the astonishment and horror of the students they found that he was dead."

Although this case might be considered as one of death due to mental shock, especially in the absence of any demonstrable pathological lesions otherwise accounting for the accident, the assumed cause is doubted by some authors. For instance Collins-Warren believes that the death was due to simple heart failure from fear and excitement. Practically, however, any such explanation does not disturb the direct causative influence of a powerful mental impression in producing instant death.

Whatever may be said of the probability of mental shock by itself causing death, there is no doubt concerning the numerous and complex influences exerted upon the different organic functions by powerful emotions. In fact these phenomena are so frequently witnessed in a more or less modified shape, that they become a part of general and every-day experience. When these impressions are added to those of injuries proper they have a very marked effect upon the prognosis, for good or evil, according as the emotions are of an inspiriting or depressing character. Victorious soldiers suffer less from shock than do those that are demoralized. Despair at the prospective loss of a limb, apprehension of the result of an operation, and fear of death markedly determine the gravity of shock.

The most severe form of mental emotion which may be considered in the light of mental shock is that of sudden terror. The phenomena of this condition closely resemble many of the leading features of severe shock from injury. The patient becomes pale, the respirations hurried and shallow, the body is bathed in perspiration, the heart palpitates, the muscles relax, the pulse becomes feeble, the eyes staring and motionless, and the sphincters relax. Under such circumstances the various functions are profoundly affected. In the nursing woman the secretion of milk may either be entirely arrested or be so altered in quality as to seriously affect the nursling. Cases are on record in which death has occurred to the latter, also others in which fright has resulted in more or less persistent aphonia, and still others in which deafness has followed.

The secondary effects of mental shocks are generally more lasting than those following physical injuries. One strong reason for this is that the original cause is apt to repeat itself in a more or less modified form for indefinite periods. Memory and association are two powerful elements in this direction, and conspire in unguarded moments to invite depression of spirits or even absolute despair. It is frequently noticed that such recurrences are during the half-wakeful hours of early morning when every other counter-active impression is forced into the background and the original cause stands out in the hard and distinct outlines of an almost overwhelming calamity. One patient who had suffered a mental shock from business losses told the writer that these secondary manifestations were to him really more overpowering than the original one. This indeed appears to be a common experience, and explains many suicides during such periods. Especially are the secondary effects persistent in patients who have lost limbs by operation or have become mutilated by accident. Under such circumstances it is often necessary to tide over the periods of depression by proper mental discipline and by the administration of stimulants. With the lapse of time, however, the mental impressions become less pronounced and finally disappear. In exceptional cases some permanent physical defect results which is doubtless due to corresponding organic changes in the cerebral centre. Victims to severe mental shocks often maintain that they have never enjoyed as good health as before the occurrence of the original attack—a fact that is well substantiated by the persistence of marked neurasthenic symptoms. It is a well-established clinical fact that glycosuria is often associated with mental shock as a causative influence. Again not infrequently the mental conditions become entirely changed, transforming quiet dispositions into irascible ones, perverting high moral propensities into degenerative proclivities, and establishing morbid fears over which there is little or no personal control.

Although the secondary effects of shock are liable to follow in any cases of traumatism proper, they have been more frequently noticed and more thoroughly studied in connection with those following railroad injuries. The number of suits for damages in such cases, wherein it has been claimed that permanent impairment of health has resulted, has doubtless accounted for this, and has in consequence given an undue prominence to this special class of injuries as compared with others which are equally prolific of bad results.

The influence of intense mental pre-occupation balances chances in an opposite direction and covers also those cases in which by force of will and pluck the shock is delayed, lessened, or aborted, and the sufferer saved from immediate death, by being thus able to help himself. This is seen often on the battlefield in soldiers arresting hemorrhages from their own wounds and in otherwise caring for injured parts. The writer recalls the case of a young man, whose right hand was shattered by the accidental discharge of a gun, and who being alone at the time on a Western prairie, was able to walk a distance of fully three miles with a sufficiently firm grasp of the wounded wrist to arrest a profuse hemorrhage. In another case, that of a medical friend, both legs were crushed in a railroad accident, and while alone on the track by a supreme effort of will he administered to himself a full hypodermatic injection of morphine which reduced what would otherwise have been a severe shock to one of comparatively trifling extent.

Such instances are, however, exceptional in reference to influence of mental pre-occupation, as in the end, as we have already seen, the effects of primary shock become more intensely retaliatory in proportion to the strain of arrest. Mental pre-occupation may sometimes be so intense, as in the line of expectation of serious injury in an operation wherein the will power is exerted to its utmost capacity, that the sudden relaxation of mental tension may bring on a revulsion precisely similar to shock. This would possibly explain such a case as that related by Sir Astley Cooper in which the mere lancing of a finger without anæsthesia was followed by sudden death. The manner in which these phenomena may be accounted for may simulate that so ingeniously described by Dr. Haig in cases of death from chloroform syncope. Dr. Haig maintains that the latter are caused by a "high and rising blood pressure which overpowers the heart and produces the syncope," and always occurs after the chloroform is withdrawn and when the consequent rebound of the circulation takes place.

It is claimed by many surgeons that temperature and barometric pressure have a marked influence on the degree of shock. Hewson maintains that shock is most severe in very cold and very warm weather. Abbe, who has collected more recent statistics, does not agree with such a view, although he admits that a rapid falling barometer may be a temporary deterrent of surgical operation. The objections to particular seasons might have held in pre-aseptic days, but practically speaking, time, place, and season are indifferent considerations when backed by the more weighty counteractive ones which apply to the more perfect technique of the surgical procedures of the present time.

While shock in the great majority of cases is a condition most frequently seen in surgical practice, it is also quite common in those which are purely medical in character. Certain poisons operate in this manner when swallowed, as for instance oxalic acid, sulphuric acid, caustic potash, arsenic, corrosive sublimate, and tobacco. The shock in such cases is as sudden and alarming as after a surgical injury, and is due to a similar impression upon the sensitive nerves of the parts involved. The same may be said of the passage of a biliary or renal calculus, the sudden escape of an irritating fluid into the peritoneal cavity from a perforating ulcer of the stomach, intestine, or appendix vermiformis, or a severe case of spasmodic colic due to intra-intestinal reflexes. Although the symptoms of general shock in these so-called medical cases are virtually the same in all their leading features, and correspond with those noticed after severe injuries and profound mental impressions, there are nevertheless certain distinctive modifications and associated conditions dependent upon causative elements, as shown in the general symptoms of each lesion, which enable us to recognize the different forms. The term *collapse* is most frequently used to indicate these varied conditions and is one which more directly applies to them.

Reaction from Shock.

The symptoms which indicate nature's efforts at restoration after shock are usually described as those which belong to a general condition, termed that of *reaction*. The procedure as a whole may be considered as a reversal of the phenomena of shock.

The first significant symptoms of reaction are manifest in the improved condition of the pulse in the directions of increase in fulness, steadiness, and force, and return of a flush to the face. The respirations also become deeper and fuller, and there is a marked disposition to sigh. The repeated appearance of the latter symptom is a particularly favorable indication. Associated with this is a return of the power of swallowing. Occasional vomiting is also a good sign, indicating a reappearance of sensitiveness to normal reflexes.

A disposition to change the position by turning from the dorsal to a lateral decubitus, and a corresponding helpful movement of the limbs, are also of favorable import.

The skin gradually loses its pallor and becomes warm and assumes a healthful flush. Very soon thereafter there is a slight rise of animal temperature. The latter in its turn soon drops to its normal standard, the pulse becomes steadier, the skin assumes natural moisture, and the patient is fairly convalescent. The proper reaction is that in which all these symptoms are gradually progressive toward the favorable result, although the question of the time in which this may take place is far from uniform; some cases requiring only an hour or two, while others occupy a period of days. The time at which reaction occurs depends largely on the degree and extent of injury, loss of blood, age, temperament, and surroundings of the patient. The longer it is delayed and the more irregular its course the graver the prognosis. Cheever declares unless reaction is fairly established within eighteen hours it need not be expected.

Under certain circumstances the course of favorable symptoms may be interrupted, but when once begun they are never entirely suspended for any length of time unless the outlook becomes correspondingly grave. When the reaction becomes excessive the patient may drift into a state of excitement which may in the end cause the balance of his chances to be thrown on the wrong side. This condition is not infrequently met with in the severer form of shock and may drift into that of shock with excitement, the erethistic form described by Travers. Such is always associated with more or less grave apprehension as to the ultimate outcome, inasmuch as there may not be enough of reserve power to meet the excessively abnormal action. The absence of such power is evident in those cases that have a protracted period of shock followed by quick and inordinate reaction.

Of the two evils, deficient or excessive reaction, the latter is the lesser. It is easier, as Roberts truly says, to control force than to create it. Any extra activity may be overcome by judicious treatment, so long as the power is properly conserved to balance it.

The increased temperature of reaction generally shows itself within a few hours after the injury and has a direct relation, in the lines of cause and effect, with the original prostration. It reaches its height by progressive steps and declines gradually, entirely disappearing at the end of twenty-four or forty-eight hours. This rise of temperature is entirely different from that occurring independently of the shock, two or three days after it, and dependent on septic infection.

Prognosis.

As has been previously intimated, very many considerations are to be taken into account in estimating the chances of recovery from shock. Generally speaking, the more sudden and severe the injury the graver the prospect. Very young people are quickly impressed and as quickly rebound. The aged, although equally susceptible to shock, recover slowly. Hemorrhage is always a bad complication, as is also severe mental prostration. The same may be said of subsultus and hiccough. Nervous temperaments suffer most acutely, while lymphatic subjects are least affected. The habitual drinker is apt to drift into delirium tremens, always a bad and sometimes a fatal complication. There is no rule of relationship between the intensity of the shock and the rapidity of recovery from the same. Severe shocks are oftentimes of short duration, while apparently trivial injuries extend their prostrating effects over long periods of time. Not alone hours but days and weeks may elapse, in the latter cases, before all the various functions affected by the shock are restored to their normal standards. In some instances, as we have already seen, permanent invalidism has resulted.

In cases of severe shock the time element is one of the greatest importance. The outlook is much better in a case of prompt recovery after severe prostration than in one with a slower rebound after a comparatively trivial traumatism. The longer the delay in the complete restoration of function the worse for the patient. Savory very truly maintains that if at the expiration of some hours no symptoms of reaction appear the patient's condition is very desperate. Cheever's eighteen-hour limit for establishment of reaction must also be recollected. Sir William McCormack states: "A fall of temperature below 96° F. nearly always presages a fatal issue. It is a very unfavorable sign when no rise of temperature takes place in four or eight hours after receipt of injury."

As long, however, as there are any attempts at reaction there should be hope of recovery even after the severest injuries. With proper treatment the cases which appear to be the most hopeless at the outset may in the end prove the most successful, while contrariwise an unexpected complication or an unlooked-for incident, in what might appear to be an ordinary case, may end in sudden death.

A great deal depends upon whether or not a vital part is directly implicated in the injury. If the latter is the case the prognosis should always be guarded. This injunction particularly applies to injuries of the brain with the added symptoms of concussion or compression. In such cases there is not only the immediate danger of the shock itself with the grave complication of probable lesion to the brain, but also that of subsequent inflammation in the cerebral tissues.

Great allowance also must be made in distinguishing between the effects of what may be considered a completed shock and those of a continuous shock. In the former the blow is struck at once and virtually all the damage possible is culminated in the stroke itself. When such cases rally, if they do at all, they generally do so quickly and safely. On the other hand, the cases which may be designated as those of continuous shock are always anxious ones, inasmuch as the effects of the original injury are more or less progressively acting, and thus barring nature's efforts toward complete recovery. Instances of this form are met with in cases of extensive lacerations of removable limbs or tissues in which there is exposure of large and important nerve trunks and more or less active hemorrhage from large vessels. Under such circumstances there is oftentimes, after waiting a reasonable time, less danger in operating and relieving nature, even during the shock, than in taking the chances of her ability to effect recovery unaided.

Pathology.

The pathology of shock is not well understood, for the reason that no distinctive autopsical lesions are demonstrable. Hence any attempt at explaining the relation of cause and effect is based rather on physiological assumptions than pathological facts. Considering the complex character of the nervous system, its multiplied conditional and co-ordinate functions, and the lack of a more extended knowledge of its physiology, it is not surprising that there is much difficulty in explaining the real significance of many of the experimental phenomena upon which some of the theories of causation are based.

When we study the symptoms of shock, pure and simple, or rather shock uncomplicated by hemorrhage, we are impressed with certain striking features. There is marked and general depression of nervous power and a corresponding disturbance of the peripheral circulation. There is no difficulty in assuming, when severe injury is suddenly followed by these phenomena, that the nervous system is the main, if not only, agency of their production. From the time that this part of the subject was first studied to the present, no different view has been entertained. Every observer has made his theory accord with it and has based his experiments on the indisputability of the fundamental fact.

When shock causes instant death there appears to be no doubt that the heart is directly involved. Travers recognized such a condition and was inclined to believe that the arrest of the heart's action was the sòle cause of death in such cases—an opinion also shared by Savory, who wrote: "Death from shock, then, is the result of a sudden and violent impression on some portion of the nervous system acting at once through a nerve centre upon the heart and destroying its action."

Dr. John B. Roberts, in his editorial comments on Savory's article, also agrees with this view in the following: "Death from shock seems to occur from sudden spasmodic contraction of the heart, or from sudden cessation of the contractility of the fibres of the cardiac walls." While arrest of heart action is, in such cases, the principal and determining cause of death, we shall doubtless be able to prove after more physiological experimentation and pathological study that it is only a part of a complex process extending its inhibitory influences through other centres of vitality.

It is possible to assume in cases of severe and direct irritation of nerves supplying the heart, for instance the so-called depressor of that organ, that death can be produced from "heart shock" alone, independently of any of the associated symptoms of general shock. The direct influences of such injuries on the heart have been proven by numerous experiments upon the lower animals. The irritation of the pneumogastric nerve itself as well as its branch the depressor, has a direct effect upon the heart in diminishing the pulse beats or in arresting the heart's action altogether. Warren truly states, however, that this explanation of the causation of general shock "would not account for those cases in which the rapidity of the heart's action is increased." Again he states that "irritation of this nerve does not always produce the same changes in blood pressure, whereas in shock there is always a general and considerable diminution in the blood pressure. This theory," he continues, "does not explain the weakness of muscular action, the diminished sensitiveness and many other symptoms of shock."

The experiments made on frogs by Professor Goltz, of Strassburg, have an interesting bearing upon this part of the discussion.

In imitating the well-known influences of blows upon the epigastrium in producing shock this experimenter tapped the abdomen of a frog with light and frequent blows. Not only was there a temporary cessation of heart pulsations, but also of the respiratory movements and of muscular action. Associated with these phenomena was a marked engorgement of the abdominal vessels. The same conditions of arrests of heart action, of respiration, of muscular movements, and of intra-abdominal vascular stagnation were noticed when blows of equal force and rapidity were inflicted upon other parts of the body. Here, although the effect upon the heart and abdominal vessels was quite pronounced, we must needs, when studying shock in its general and broad aspects, take into consideration those other conditions which are equally essential in making up the history of what is really a complex pathological process. Although it might appear at first from these experiments that the heart and abdominal vessels were the principal factors in producing the phenomena of shock, a further study of the latter in their relations to well-established physiological principles easily disproves such a supposition. Notwithstanding we must admit that certain portions of the circulatory apparatus are affected differently by different forms of irritation, the entire circulatory apparatus is controlled by a common influence. "The heart and the vessels," says Mansell-Moullin, "are but co-ordinate parts of one system and may be simultaneously influenced in the same direction by a single stimulus." Why one part of the vascular system is more impressed than another it is difficult to explain. Any attempt in the latter direction can at best be founded on pure theory. In the case of the abdominal circulation there may be less tonicity of the vessels than of those in other parts of the body. It was quite natural to believe, in viewing the local engorgement of these parts, that the depressing symptoms of shock were mostly if not entirely, due to this diversion of the blood current, constituting what has been aptly styled "intra-vascular hemorrhage." If such were granted the symptoms of hemorrhage and shock would be precisely similar. This, however, is not the case. The essential element in syncope is cerebral anæmia due mostly to hemorrhage, that in shock to injury along the nerve paths. Even allowing that the contrary were true, not enough blood is thus diverted in the abdominal vessels to dangerously lower the general blood pressure. Such is proven by autopsy in cases of shock, and under similar conditions during the exposure of the abdominal contents in laparotomy. Warren, in speaking of shock in connection with such operations, is certain, from his own experience, that there is no marked change in the blood supply of the abdominal vessels. Furthermore it has been found that in those cases of sudden collapse associated with rapid emptying of the abdominal cavity, as in premature delivery, the relief of ascites and the like, the condition is merely one of brain anæmia rather than of any marked change in the blood supply to the abdominal vessels. A similar fact is noted by Horsley as the result of autopsies. Physiological experiment has gone still farther toward the settlement of this question. The division of the splanchnic nerves which causes reflex vaso-motor paralysis of the abdominal vessels and consequent stagnation does not produce the symptoms of shock. On the other hand Groeningen found the abdominal vessels empty in rabbits dying from shock. Tappeiner proved that the abdominal blood-vessels of the rabbit, covering as they do a relatively large area, could not hold more than sixteen per cent. of the total amount of blood of its body, and Müller, quoted by Moullin, showed that as long as the vaso-motor nerves retained their power the total amount of circulating blood might be halved or doubled without producing any appreciable difference. The same result was attained after section of the splanchnic nerves and the consequent stagnation of blood in the abdominal vessels. It was also found by Müller under such circumstances that the fall of "arterial tension was never equal to and in the majority of cases not half so great as that which must occur in a case of hemorrhage, if the symptoms are comparable in their severity to those of an ordinary case of shock." Further than this, according to Moullin, animals in which both splanchnics have been severed have recovered, "the local centres assuming the function which the medulla and spinal cord are no longer able to carry out, and the vessels gradually resuming their calibre and tone." It became evident from these experiments that the visceral congestion, far from being the principal causative factor in the production of shock, was but an accessory condition, or rather part of a general process in which the whole vaso-motor system was involved. In no other way could the marked lowering of blood pressure, one of the significant phenomena of shock, be rationally explained. Such a theory was gradually evolved from the combined studies of Fischer, Schneider, Falk, Sonnenberg, and others. It, however, became apparent to Moullin that many of the constant phenomena of shock could not be explained on the basis of this vaso-motor theory alone, and he thus expresses himself on the subject: "It has been shown beyond the shadow of doubt that, as a consequence of injury, the molecular motion which constitutes nerve force may be interfered with, perhaps even interrupted, in certain centres that control the heart and the vascular system; may it not be that the paralysis of motion and sensation, and the impairment of reflex action, instead of being merely secondary effects produced through the agency of the circulation, are also due, wholly or in part, to a similar interference with the molecular motion in other centres? and that shock is to be regarded as an extreme and general manifestation of that inhibition with the power of which as regards a few organs physiology has made us acquainted?" In other words, it is not only likely but more than probable that all the reflex centres are involved in any injury associated with general shock. Such a view is not only broad enough to explain all the varied and complex conditions of shock, but is in every way a rational one, and in accord with our present comparatively limited knowledge of the varied functions of the nervous system. Although syncope, as such, resembles shock in many respects, it is essentially different from it in the causes which produce it. The latter are associated with weakness, usually from loss of blood, and all the symptoms are referable to such a condition. In shock, however, there is a state of prostration brought about by reflex action merely. When they coexist, as is often the case, one condition intensifies that of the other and proportionately adds to the gravity of the case.

The post-mortem examinations of shock show no distinctive lesions. The gross appearances are merely those that are seen in cases either of sudden death or of death from exhaustion. The heart may be arrested in systole or diastole. In one case the organ may be pale and empty, in the other distended and the right cavities more or less filled with blood. The brain may be congested or anæmic. The lungs are usually congested and the secondary condition of œdema is generally manifest. The abdominal organs, contrary to what might be expected, are not always congested. In three cases observed by Horsley, and to which allusion has been made, the "mesenteric vessels were not markedly full at all." The blood is dark in color and partly or wholly fluid in consistency.

Diagnosis.

Ordinarily the diagnosis of shock is not a difficult one. The suddenness of the attack of prostration after injury or other exciting cause is its leading characteristic. In this respect it closely resembles the ordinary attack of syncope, from which it clinically differs only in duration and degree. Travers says: "A fit of syncope and the recovery from it present an epitome of the phenomena of shock." It is generally agreed that syncope is due to an anæmic condition of the brain, while shock is attended with a general diminution of blood pressure in which the brain is not directly concerned. Hence unconsciousness is a leading feature in the former and a partial, accessory, and incidental one in the latter. Syncope is always of short duration, lasting but a few moments, and is generally relieved by the recumbent posture alone.

Another condition with which shock can be confounded is that of concussion. In the latter, however, we have a combination of shock with the history or objective evidences of brain injury. In concussion there is insensibility, contracted and irresponsive pupils, but the leading distinction is to be found in the character of the pulse. Fischer maintains that in brain injury the pulse is always full and slow, while in shock it is small and irregular.

The symptoms of fat embolism very often resemble those of shock. As this accident is due to a plugging of the capillaries of the lung by oil globules from torn adipose tissue or crushed bone marrow escaping into the general circulation, considerable time must be allowed after the injury for the manifestation of the process. As explaining also the usually fatal termination of a pronounced case, deposits of fat are also found in the capillaries of the kidney, brain, and other vital organs. Fat embolism generally occurs from thirty-six to seventy-two hours after the injury and seldom if ever within twelve hours; while shock follows the injury itself. In the former, also, fat appears in the urine, and for obvious reasons dyspnœa is a prominent and urgent symptom, alternating with or accompanying irregular respirations. There is sometimes hæmoptysis, and not infrequently convulsions and paralysis, which are often bilateral in character.

Acute septicæmia so often associated with shock, and so frequently causing, as in fat-embolism, the symptoms of so-called secondary shock, appears even later. The symptoms of infection are high fever, delirium, and diarrhœa, and although the onset may be strikingly sudden, there is always a given interval allowable for septic absorption.

Prophylaxis.

The prophylaxis of shock has reference more particularly to the prevention, mitigation, or control of conditions associated with surgical operations. It concerns the preparation for the operation, as well as the anticipation of dangers to the patient during its performance. A primary object is to reduce all sources of prospective and existing irritation, whether mental or physical, to a minimum. Whenever an operation is proposed the greatest hopefulness of a successful result should be given to the patient. When time allows, every advantage should be taken to increase his vitality and stimulate his resistance. The secretions should be regulated, a proper amount of sleep insured, and in some cases preliminary confinement to bed has an excellent sedative effect. Most surgeons choose the morning hours for operation, claiming that vital resistance is then at the highest point. This is based evidently on the assumption that the patient has been refreshed by a good sleep on the previous night. The heat of the body is to be carefully maintained before and during the operation. A preliminary hypodermatic injection of morphine and atropine is of great advantage in steadying the heart, quieting irritation, and markedly lessening the period of etherization. Digitalis is sometimes indicated when the cardiac pulsations are feeble. For a similar reason from $\frac{1}{20}$ to $\frac{1}{10}$ of a grain of strychnine is administered at the beginning of an operation. Lange recommends, especially in long and more or less bloody operations, the rectal injection of warm water with claret as soon as anæsthesia is established. This injection may be repeated at intervals, thus allowing time for absorption. "It is not so much the stimulant which helps the patient as the amount of fluid he takes into his vascular system," thus anticipating hemorrhage and balancing blood pressure. A simple rectal injection of warm water often answers the same purpose. The efficacy of irrigations of warm sterilized water in laparotomies in mitigating shock is attested by the experience of every laparotomist, while the sudden cooling of the abdominal contents emphasizes by disastrous consequences an opposite condition.

A simple method of anticipating the effects of shock during operation is that based upon the experience of those surgeons who operated before the time of anæsthesia and more recently advocated by Dr. Stephen Smith. It consists in the preliminary partial intoxication of the patient by means of whiskey, rum, or brandy in suitable quantities. There is no doubt, if the patient's stomach is not disturbed, that he is virtually "well braced" for the ordeal of operation, and is placed in a condition in which his sustaining power is exerted to the utmost. Dr. Smith asserts that under the circumstances the pulse remains full throughout even long operations, a comparatively small amount of ether is required, and reaction from the operation is prompt and efficient.

Large doses of quinine are regarded by Drs. Easley, Dow, and McGuire as having special prophylactic properties. It can hardly be, however, that this drug in itself has any marked or special action upon the reflex centres, or indeed upon blood pressure generally, but rather operates indirectly in controlling the depressing effects of malarious poison so common in various districts of the South.

Treatment.

Much that has been said under the head of prophylaxis of shock may be made to apply to its treatment. In fact the bearings are virtually the same, whether they refer to prospective or existing indications for relief. Generally the latter can be considered under one or other of three headings, viz., rest, restoration of animal heat, and stimulation with nourishment.

Too much stress cannot be laid upon the importance of the first of these. Rest is in every sense the contrast of the irritation producing the shock. Every leading symptom of the condition emphasizes the necessity of absolute quiet. The patient himself shows by his apathy, indisposition to voluntary movement, and other evidences of low vitality, nature's imperative demand in these cases. So much so, indeed, that unnecessary movements are not only painful but oftentimes dangerously prostrating. Hence the less he is disturbed directly or indirectly the better the chances for a prompt reaction. The rest is not so much a question of kind, so long as we bear in mind the absoluteness of its degree. Essentially it consists in the removal of all existing and possible sources of peripheral irritation, whether subjective or objective. Every active or even passive movement, under the circumstances of shock, is an expenditure of nervous force and a consequent drain upon a dangerously depressed vitality. What would be an ordinary exertion in a state of health might cause fatal exhaustion during the existence of shock. All the vitality in severe cases is diverted in the direction of a strained endeavor to conserve the necessary power for sustaining a flagging heart. Very often in severe cases the mere removal of the patient from his bed to the operating table is fraught with great danger. It is sometimes a question also, whether or not the too hasty removal of the collapsed patient from the table, in fear of a merely technical reproach, does not invite in the end the very result so desirable to avoid. The best chances for reaction under such circumstances are either to convert the bed into an operating table or the latter into a couch, upon either of which the patient may, if needs be, rest absolutely undisturbed for hours.

The well-known influence of rest in the relief of pain is another strong argument upon the point at issue. This result is not only obtained by quieting all sources of local irritation, as has been so well shown by Hilton, but by placing the entire nervous system in a properly receptive state for the restoration of its lost energies.

Under the head of rest must be noticed the beneficial influences of sleep. If the patient can sleep he doubles the effects of simple rest. The worst cases of shock are those that cannot sleep. Persistent insomnia is always a bad sign and generally portends a fatal issue. Incidentally it may be here remarked that opium, aside from its beneficial effects in stimulating the heart, is the most efficient drug for producing sleep.

Posture as conducive to rest is an important consideration. The patient should lie upon the back, as this requires the least effort; and in order to maintain an equable circulation the head should be level with the bed. Generally it is better, in following out the same indications, to raise the foot of the bed.

The next in importance, on general principles, is the speedy restoration of animal heat. Instinctively the patient shivers and either by wistful looks or urgent words asks for blankets and warmth. Besides this, the application of artificial heat by means of hot air, or hot water in rubber bags or bottles under the cover, is always indicated. Care should be taken, for obvious reasons, that the patient should not be burned in the commendable zeal to make him warm. So many accidents happen under such circumstances that too much watchfulness cannot be enjoined. It is an excellent plan to have a heating apparatus attached to the operating table for use in bad cases.

For the ready application of dry heat, in the absence of the usual apparatus, an elbow of stove-pipe or tin gutter with one extremity under the bedclothes and the other over an alcohol or other lamp, may answer in emergencies. Hot bottles wrapped in cloths are, however, always readily available.

Heat can be applied in other ways locally and generally to follow similar indications. Dr. Hunter advises that the entire body be immersed in a bath with a temperature of 98° F., gradually increased to 110° F. It would seem, however, that the good to be thus obtained would be more than offset by extra handling of the patient. Dry heat can be advantageously applied to the precordial or epigastric regions, or the same parts can be stimulated by sinapisms. Large rectal injections of hot water are also of service in conjunction with the means already noticed.

As regards stimulation, which may be made to include all other forms of medication, one injunction of the greatest importance is not to attempt to do too much. The patient is not in a condition to tolerate much fussiness on the part of his attendants. He can bear very little, and his helpers should forbear a great deal. It is well in this connection to agree with Paget in the fact that "there is perhaps no case in the management of which the courage to do little is more needed. Great energy of treatment may do great mischief."

Of all the stimulants whiskey or brandy is the best. Its effects, save in exceptional cases, are always prompt, certain, and decided. When retained by the stomach it acts with greater advantage than when employed in any other way. It must be given, however, with circumspection, else the weakened stomach gets more than it can absorb and vomiting results. A good rule is to give the stimulant in small quantities often repeated. In proportion as the pulse gains strength and animal heat returns, the quantity and frequency of the dose should be lessened, otherwise reaction may become excessive. When the stomach cannot retain the stimulant it may be introduced *per rectum*, half an ounce to an ounce at a time, largely diluted, or it may be given hypodermatically in quantities from half a drachm to a drachm every ten or fifteen minutes. The enemata prove especially useful when administered hot, as, aside from the benefit of direct absorption, there is another of heat stimulation.

Next to whiskey or brandy as a stimulant comes coffee. Cheever believes that there is no better stimulant than a mixture of brandy and coffee when it can be retained by the stomach. When the latter is not the case, warm coffee alone or mixed with brandy can be given by the rectum.

Under the head of medicines for the treatment of shock, opium holds the leading place. It has the double function of a general sedative and heart stimulant. In both these capacities it renders good service in different stages of shock. In sufficiently large doses it is capable of quieting the most severe pain. Being thus able to remove one of the main causes of shock, it properly prepares the way for its other valuable service. Its use in both directions is indicated only so long as depression is present. All other indications point to its discontinuance. If its administration is persisted in, notwithstanding, harm may come by courting a too speedy and too violent reaction.

It is often surprising to note the great tolerance for opium in both of the conditions named. The large doses are graduated to the amount and urgency of the pain. Any quantity of the drug is safe so long as it balances the degree of pain. In other words, the drug must be used in sufficiently large doses to control the pain, allowing sufficient intervals of time for effects between repetitions.

As a rapidly diffusible general stimulant carbonate of ammonia is considered to be most valuable. It can be given in doses of five grains in a suitable menstruum every half-hour. Richardson states that it has a quality of maintaining the fluidity of the blood, thus preventing heart clot. An agreeable form of administration is the officinal spiritus ammoniæ aromaticus in drachm doses suitably diluted with water. Sulphuric ether is often substituted in the same dose mixed with aromatic water.

The hypodermatic injection of digitalis is often of great service as an adjuvant to the stimulants already mentioned. It may be administered in fifteen-minim doses of the tincture repeated every fifteen minutes for the first hour, after which due allowance must be made for the cumulative tendency of the drug. It is generally desirable to add one one-hundredth of a grain of atropine to the first injection.

Verneuil as long ago as 1877 recommended the hypodermatic injection of ether in cases of shock, and since that time its use has become quite common in operating theatres, many surgeons preferring its stimulating action to that of brandy similarly employed. It may be administered very freely, thirty minims every five or ten minutes until the patient rallies.

Hypodermatic injections of atropine in doses of $\frac{1}{80}$ to $\frac{1}{50}$ of a grain are oftentimes of signal service. Bradner, after an experience of twenty-five years, believes this drug to be the best of all heart stimulants. Eggleston states that it not only increases the number and vigor of the heart beats, but the area over which the pulsations are distributed is enlarged, thus influencing in a marked degree the peripheral circulation and the consequent elevation of temperature. It is considered by him in such a view of special value in the shock from lightning stroke.

The tincture of belladonna is sometimes used internally in half-drachm doses every hour, also with the view of stimulating heart action and aiding contraction of the arterioles, but it should be regarded only as a substitute when atropine is not available.

The promptly beneficial action of the inhalation of nitrite of amyl in the hands of the writer in cases of threatened chloroform narcosis has induced him to try the remedy in shock proper. Good effects may be obtained in cases where something must be done quickly or left undone. He has used the remedy with good effect in the latter condition. He would consider it specially indicated in threatened cardiac failure when it was necessary to stimulate the cardiac muscle, and rapidly revert the force of the blood current from the cerebral to the peripheral regions. It must be borne in mind that such effects can be produced by a simple whiff or two of the vapor. On no account should the administration be pushed beyond this, as the effect is not only practically instantaneous, but is liable to increase after the vapor is withdrawn. In full doses the drug produces rapid heart action, great flushing of the face, diminished arterial tension, muscular relaxation, and gasping respiration. In smaller doses administered with due caution it would appear to act indirectly as a quick, powerful, and diffusible circulatory stimulant.

Various other drugs have been suggested as useful in shock, but none so far has won an important place in the recognized list of well-tried therapeutical remedies. Among such may be mentioned hyoscyamus, chloral, and the various coal-tar products.

Stimulants, however, are of use only as temporary expedients in pressing emergencies. They may start the fire, but the real fuel must be supplied by subsequent nutriment. Hence when the pulse becomes fuller, large and well-warmed enemata, but better still plain nourishing fluids by the mouth, may be given. Milk is the simplest and best food for the stomach, while beef tea mixed with egg, or peptonized beef juice, answers best per rectum. Milk and other liquid foods should be given slowly and in small quantities at a time in order to insure proper admixture with the saliva, thus giving the natural start to the gastric digestion. When nausea is present small quantities of hot beef tea per rectum answer an excellent purpose.

The intense thirst which exists more especially in cases that have suffered from severe hemorrhage must be relieved as cautiously as is consistent with giving all the water the patient can absorb. The writer has never seen harm come from drinking any reasonable quantity of water when the latter could be retained by the stomach. In the intervals pellets of ice may be allowed to melt in the mouth. It is always better to look up to nature than to bow to a theory. When in the severe cases of hemorrhage the systemic thirst is intolerable, when the stomach is too irritable to retain water, even in small quantities often repeated, the saline solution recommended by Mikulicz may be injected into the median basilic vein or in the cellular tissue of the abdomen or other available region. This solution is prepared after the following formula: sodii chloridi, ʒ iss.; sodii bicarbonatis, gr. xv.; aquæ destillatæ, Oij. Mikulicz advises that from one to four pints, warmed to 100° F., be slowly injected, the pulse and respiration being carefully watched. Roberts believes, and with good reason, that saline solutions thus given will be more promptly efficacious in quenching thirst than water by the mouth.

Sir William MacCormack and other authorities suggest that it would be proper, when the heart is overcrowded with blood during shock, and the jugular veins gorged, that blood be drawn from the latter vessels. It is assumed that by such means the general blood pressure may be quickly and directly impressed and thus the distended heart be relieved. The condition apparently indicating such a line of procedure is rather an effect than a cause of heart distention. As Moullin very properly suggests, "the heart is distended because it is paralyzed, not paralyzed because it is distended."

In those cases in which there is great cardiac feebleness from lack of volume in the blood current, as in syncope from hemorrhage, bandaging the extremities is of great benefit in keeping the intra-abdominal and intra-thoracic vessels sufficiently full to enable the heart to act properly. In case there is not a quick response the saline intravenous injections are urgently demanded.

It is claimed in cases of collapse that the phrenic nerve may be usefully stimulated by electrodes placed along its course on the neck and over the epigastrium. This, however, is always a forlorn hope, and, except for the show of doing something when a patient is past help, is of no use whatever. It is often tried and as often fails.

When reaction becomes excessive the indications, as pointed out by Travers, are to support and increase power as well as to moderate and reduce action. Stimulants are discontinued, excitement is modified by anodynes, but most important of all, every possible nourishment must be obtained by food administered in proper quantities and at proper intervals.

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SEASICKNESS—NAUPATHIA.

Seasickness is an affection which seldom finds more than cursory mention in systematic medical treatises. Nevertheless, many thousands of human beings suffer from it daily, and some so seriously as to demand medical interference for the relief of distressing symptoms or to prevent permanent impairment of health or possible danger to life. A morbid state, whose range is from an evanescent malaise to the most profound lethargy, from mere dizziness and headache to coma or collapse, and which, as Pellarin has said, is capable of imposing the sacrifice of all a man holds dear—his ambition, his most natural affections, life even—deserves the careful attention of the physician. Its English designation appropriately indicates it, as do its equivalents, *Mal de mer* in French, *Seekrankheit* in German, *Zeeziekte* in Dutch, *Mareo* in Spanish, and *Male di mare* in Italian. Its customary nosological title, *Nausea marina*, is a misnomer, since the Latin adjective is a needless tautologic postfix to the Greek original, *ναυσία* from *ναῦς*, a ship, which evinces the early association of its prominent symptom with its usual maritime cause. For purposes of international medical statistical nomenclature *Naupathia* is the preferable term. Irwin proposes *Kinetia* as a more rational name, since it includes all other similar forms of motion-sickness.

Although seasickness is an absolutely avoidable infliction, since no one will be affected by it who remains on shore, and notwithstanding scarcely five per centum of the human race, who do go afloat, are wholly exempt from it, the love of novelty and desire to travel are so impellent, that the number of its victims is no more lessened than is the population of the earth retarded by the pains of childbirth. In fact, the two conditions are alike in that while both are, at the time, bewailed in extravagant terms of terror and abhorrence, both are commonly so soon forgotten by the great majority of sufferers that the *peine forte et dure* is again and again voluntarily and deliberately encountered. Occasionally, however, families have been permanently separated through the unconquerable dread of the sea inspired by one voyage, and instances are not rare of officers of the naval and merchant services who have been compelled to abandon their vocations by reason of constitutional unfitness. Cato declared that one of the three things he regretted having done was that he went by sea when he might have travelled by land.

Symptoms.

The indications of seasickness are unmistakable. A person apparently or actually in the best possible physical condition embarks on board a vessel of any kind, and sometimes even before the anchor has been lifted, but usually not until a longer or shorter time after getting under way, exhibits a train of symptoms of varying intensity, which are, however, recognized as due to the change of habitat from terra firma to a contracted, unstable domicile afloat. No class, condition, or age is exempt, although very young children are seldom affected, the susceptibility increasing from the second to the eighth year of age, when they suffer like adults. Bronsmiche, *Médecin principal* in the French navy, quoted by Martialis, stated that on a transport to which he was attached, during a severe gale, when even old sailors were affected, forty officers on board were all sick, while thirty-two children from three months to four years old enjoyed complete immunity. Neither is it peculiar to man, monkeys and quadrupeds, especially horses, cattle, and dogs, succumbing, cats less than other animals, while domestic fowl and other birds are often too sick to swallow the food they feebly attempt to pick.

Among human beings, the degree of indisposition has a wide range. While perhaps five in every hundred persons are practically immune, twenty-five per centum will be but little sick, sixty per centum a great deal so, though for a comparatively brief period, while the remaining ten will be distressingly ill, one or two of these, if the weather be cold and stormy, the passage long, and their prior physical condition bad, becoming critical. The seasick may, therefore, be considered in three principal groups, as moderately, considerably, or severely affected.

Ordinarily the first evidence of indisposition is a feeling of general malaise, an indefinable nervousness, and discomfort or confusion about the head—a sensation of unsteadiness, variously described as lightheadedness, giddiness, or dizziness, soon amounting to decided vertigo, and attended with vague frontal headache, pain in the temporal regions, or a sense of tightness or constriction in the back of the neck and disposition to stretch and yawn, followed by irresistible drowsiness. Some never go beyond this, a hearty meal, a cup of coffee, or a sound sleep dissipating the uncomfortable sensations, and many old seafarers, who would resent the imputation of seasickness, experience, during very rough weather when the temperature is low and the sea choppy, this mild form of cerebral disturbance, which, however slight or severe the attack, is the one invariable concomitant of this affection. In aggravated cases it not unfrequently outlasts recovery, continuing as a lingering souvenir of the voyage, women especially who have suffered grievously declaring for many days after that they still feel the motion of the vessel.

Not uncommonly with the giddiness and headache, the temper becomes irritable. The victim shuns association, either resenting the banter of old voyagers or morosely indifferent to it and low-spirited. The face flushes and pales alternately, the light is painful, the pupil contracting to exclude it; there is ringing in the ears, and an exaggeration of the sense of smell, that makes customarily unnoticed odors, as that of a cigar, or the otherwise agreeable smell of cooked food, revolting. At the same time, there will probably be epigastric uneasiness, "sinking at the pit of the stomach," and sense of emptiness with a craving for food, if the general health be robust, rather than the usual loss of appetite. If this be gratified judiciously by a bowl of hot soup or a glass of kumyss, the nervous equilibrium will be restored, and the cerebral hyperæmia and disturbed sensory impressions relieved, so that the case will never exhibit the decided nausea which has given its recognized technical name to the condition. The fortunate few whose indisposition is limited to this are those whose appetites become voracious, whose step recovers its spring, and who promenade the decks rejoicing in the invigorating and exhilarating influence of the ocean air.

With the far greater number the vertigo and epigastic discomfort increase, anorexia is developed, and soon after nausea which is aggravated by the unaccustomed sights and smells until vomiting occurs, followed by relief of all the unpleasant feelings, which, however return again and again after brief seasons of rest and sometimes sleep. The symptoms of nervous asthenia at the same time become more prominent. Vertigo is followed by faintness, mental depression, and hebetude. The sufferer answers laconically if at all. Respiration is labored and sighing. The circulation becomes slow, weak, and sometimes intermittent, and as a consequence the extremities are cold, the nails blue, "chills creep up and down the spine" alternating with flushes of heat, with no actual rise of temperature. With this there is loss of muscular co-ordination, similar to that of drunkenness, the gait is staggering, the legs bend under the weight of the body, and the unfortunate falls helplessly into a seat and refuses to make an effort to rise. A few, with naturally strong will power, protest to the last that they are all right, although their changed physiognomy betrays

YEAR.	Admissions for "nausea marina."	Total number of days sick.	Average duration of each case.	Transferred to hospital.
1879	28	92	3.28	1
1880	28	76	2.71	1
1881	34	102	3.00	3
1882	73	224	3.06	..
1883	62	209	3.37	2
1884	45	213	4.73	2
1885	40	97	2.42	..
1886	30	87	2.90	..
1887	8	23	2.87	..
1888	26	74	2.84	..
1889	21	59	2.81	2
1890	69	163	2.36	..
1891	54	175	3.24	..
1892	58	187	3.22	..
1893	85	214	2.51	1
Aggregate	661	1,995	....	12
Annual average	44	133	3.00	..

During this period, there were annually borne on the rolls of the navy, as the effective force afloat, the names of from ten to nearly eleven thousand officers and men, of whom perhaps one thousand a year represented young men and boys from civil life—enlisted for the first time as landsmen, clerks, mechanics, apprentices, marine recruits, and newly-appointed cadets at the Naval Academy, só that a maximum of less than five per centum of these ever fell under the cognizance of the medical officers and became matter of record as victims of seasickness; and though they must have belonged to the class of seriously affected to have been placed on the sick list, the average duration of their inability to do duty was only three days, and in fifteen years but twelve required to be sent to a hospital, and none of these terminated fatally or was invalided from the service.

Dutton, who, though himself an old sailor, admits, "I have never mastered my own tendency to seasickness," referring to his memoranda of many thousands of cases, says: "I have never lost a case, but among my notes I find two who nearly succumbed to the dreadful malady—one an Italian dentist, who was ill all the way from London to New York, who did not keep anything on his stomach longer than ten minutes during the ten days' passage; the second a Scotch lady who was sick with hardly an interval from London to New Zealand. We arrived at Wellington in forty-two days after a fair passage, where we stayed some days, and where she picked up a little, but on the voyage from Wellington to Port Chalmers she was as bad as ever." De Rochas reports the case of a nun, who was seasick every day of a six months' voyage to Australia, and who was reduced by the persistent vomiting to the last degree of emaciation, and I myself saw a Dutch nun, on a twelve days' passage to Amsterdam, when the sea was smooth as a mill-pond all the way across, and there were no other cases among the five hundred persons on board, who might have died from inanition and nervous exhaustion, but for careful treatment and compulsory feeding.

Fordyce Barker, however, relates his experience of three fatal cases, one a young physician, his former pupil, who sought to recuperate from the exhaustion of overwork by a fishing excursion of some weeks to the Banks of Newfoundland, and who, after four weeks of uncontrollable vomiting, died, two days after landing, delirious and exhausted from inanition; a second of a young lady, who died on the passage from Havre to New York, and whose death was attributed by Dr. A. H. Smith, the surgeon of the steamship, to exhaustion, unquestionably the result of the seasickness alone; and a third, whom he saw in consultation, who died on the fourth day after arriving from a rough passage of sixteen days from Liverpool, who had been unable to retain anything on the stomach during all that time.

Gouin, on board *L'Éperlan*, had under his care a young marine artillerist, returning from the Gaboon, who had been greatly debilitated by long residence in the unhealthy climate and by numerous attacks of paludal fevers, who died after nine days of uninterrupted vomiting. The emesis became bloody, the prostration was extreme, the skin was cold and clammy, frequent attacks of syncope supervened, during one of which he died. A young native of Senegal, Gouin states, died on board *L'Australie* under similar circumstances.

Forget relates a case of encephalitis consequent upon seasickness; Ménard a fatal case of gastro-enteritis; Guépratte a death from hematemesis; Fonssagrives one from rupture of the stomach; and Andreux had four deaths among four hundred passengers, many of them chronic invalids, returning to France with constitutions completely broken down by tropical diseases.

Causation.

The rationale of the processes which set up the vertigo, nausea, and prostration, constituting the several phases of seasickness, has been a matter of speculation by the various authors whose works are indicated at the close of this article. A dozen or more theoretical explanations have been propounded as to the precise manner in which the motion of the vessel disturbs the organism. The latest authorities agree in placing the affection among the neuroses. Skinner, whose admirable essay appeared a little over a year ago, regards it especially as a neurosis of the great sympathetic—the movements of the vessel primarily affecting the sensorium, or mechanically displacing and straining the visceral organs, or altering the pressure of the column of blood on the containing vessels and thus inducing a reflex inhibitory influence on the cardio-accelerator and vaso-constrictor centres, whose paresis enfeebles the heart's action, dilates the sanguineous vessels, and lowers the general arterial blood pressure—the last being one of the starting-points of what he terms "a veritable vicious circle, which explains the tenacity of the affection and its resistance to divers methods of treatment." Van Valzah of New York, one of the very latest and most logical writers on the nature of seasickness, defines it to be essentially and primarily a disordered sense of equilibrium and of space, inducing a sensory form of vertigo, due to hyperæmia and instability of the sensory and sympathetic nerve centres, and the exciting cause continuing to act, the resulting anæmia of the brain and cerebro-spinal irritability explain the mental depression, gastric symptoms, and nervous exhaustion and prostration which ensue with increasing intensity. Bénard, one of the later French theorists, recognizes the *mal de mer* as a neurosis, a simple malaise when mild—a sympathetic nervous vertigo accompanied with gastric disturbance when severe—due to the perverted sense of space or equilibration, which he locates in the semicircular canals.

Vertigo is unquestionably the constant phenomenon in whatever case of seasickness, and disturbed equilibrium the constant precedent condition, but any irregularly moving conveyance, besides ships, occasions vertigo, as expressed in the popular phrase, "It makes me seasick," during a ride in a railway car over deep grades and around sharp curves, or in a stage-coach or calèche hung on high springs, or on the back of a trotting camel. The proposed military dromedary corps in the French army was found impracticable because of the sickness and disability of so many of the soldiers induced by the peculiar gait of the animal. The gyrations of the waltz, especially if continued without reversing, the motions of the swing and see-saw, looking down from high altitudes into chasms, and ascending in a balloon, all act by disturbing the sense of equilibrium and occasion vertigo, headache, nausea, vomiting, and syncope as in seasickness. The same symptoms are also induced by the wave-like undulations of earthquakes in countries where these occurrences are frequent, among those unused to them, and have been wrongly attributed to fear. Old writers, Plutarch among them, did indeed ascribe the phenomena of seasickness to emotional disturbance, of which fear was the incitant, and Guépratte in support of this possibility tells the story of two shipwrecked whalers, rescued by a French vessel, who subsequently became seasick when doubling Cape Horn; but while mere dread of the sea may act as a predisposing influence in a few individuals of timorous disposition, and the recollection of their peril may have had this effect in the two whalers whose nervous systems had been shattered, the effective cause will always be found in actual or imagined disturbance of equilibrium. A distinguished surgeon in the United States Navy, formerly associated with me on duty, who, although he had passed half of his twenty-five years of service at sea, was always a great sufferer from seasickness, assured me that he could at any time excite in himself feelings of nausea, by recalling occasions and circumstances of former attacks. Charteris quotes Henry Ward Beecher as relating how "many years after his first voyage across the Atlantic, he heard some sailors in a Brooklyn dock singing the same old 'chanty song' that he had heard when ill at sea, and that the mere listening to it produced the creepy feeling of seasickness;" and tells another striking instance of the effect upon the sensorium of the association of ideas in reawakening the peculiar evidences of this condition, which he observed three years before, when, as he was standing on the deck of the little steamer which was about to start from Penzance in Cornwall for the Scilly Islands, a cab drove hurriedly up to the pier and from it alighted a lady and a gentleman, the former of whom was so affected by the mere sight of the steamer that she became pale, staggered, and gasped for support from dizziness, and no persuasion could induce her to go on board. Martialis also narrates, in illustration of how easily the sense of equilibrium may be affected through sight alone, the case of a lady on board a naval dispatch boat at anchor in a closed dock at Havre, where the vessel was absolutely motionless, who got seasick from merely watching a lamp swinging in the wind from an open skylight—an effect similar to the sensations induced by the use of too powerful eye-glasses upon those not accustomed to such aids to vision. Dr. Macauley of the United States Army tells how Senator Morrill was relieved of his seasickness by checking the swaying of "that confounded lamp," which fascinated him so that he could not keep his eyes off it, and which was the only thing that could have disturbed his sense of equilibrium, since he had been carefully swung in a cot, steadied by guy-ropes, in the hurricane-deck house of an old sidewheel transport, where he could neither see the water, feel the motion of the vessel, nor smell any of the ordinary disagreeable odors of passenger steamers. Jobard knew a lady who could not even look at a marine painting by Gudin without becoming seasick.

The sense of smell, like those of sight and hearing, may be the sole starting-point in the chain of phenomena ending in nausea and other indications of seasickness, and I am able to contribute from my own personal experience a notable instance of the disturbing influence of such a single sensory impression. After more than twenty years' service in the navy, during the whole of which time I had never been seasick in the slightest degree, I took passage with my family in a coasting steamer at Baltimore for Savannah, and noticed as soon as I got on board an intolerable stench of what seemed to be bilge-water, which grew worse after we got under way and continued to increase as we steamed down Chesapeake Bay, because, I took for granted, of the agitation of the vessel by the engine. Instead of gradually diminishing as I expected, it became more and more insupportable, until a qualm of unexpected, and to me entirely novel, nausea caused me to rush from the table and lose my first meal. A cargo of bone phosphate fertilizer stowed under the saloon had done what the most violent typhoons in the China Sea and West India cyclones had not accomplished.

The determining influences in most cases of seasickness are complex. Previous illness, antecedent imprudences in diet, physical and mental fatigue, the interruption of accustomed habits and surroundings, and the change to entirely novel conditions of life increase the susceptibility, which is naturally excessive in some individuals, to the mere sensory impressions produced by the unstable horizon, the moving waves, the roll of the vessel, or the smell of the machinery, to which they might otherwise be indifferent. Ordinarily no other cause is necessary to explain the occurrence of seasickness than the motion of the vessel, and the degree of the indisposition will be in proportion to the amount and irregularity of that motion. Many of those who soon accommodate themselves to the slow, easy roll, when the wind is abeam or on the quarter and only a long regular swell disturbs the surface of the ocean, are overcome when the sea is short and choppy, or a head wind and head sea cause the vessel to plunge and pitch as well as roll. The majority of writers on seasickness have erred in attempting to explain the condition as the result of an invariable exciting cause operating in one direction. Darwin believed the vertigo to be always due to the disagreeable affection of the sight by the continual mobility of surrounding objects, and that the brain thus impressed sympathetically affected certain other apparatus. Barun made the optic nerve the seat of primary irritation caused by the apparent vacillation of everything around the vessel. As recent an author as the late Dr. Graily Hewitt, arguing from his own personal impressions, thought that visual disturbances were at the bottom of every case of seasickness. That such disturbances may cause seasickness has been abundantly demonstrated, and Macauley claims to have made people actually seasick on shore by imitating the motion of the ship with the two hands, the thenar eminences together, and slowly swaying and opening them like a shark's jaw. "In a perfectly smooth sea once when a boy," he says, "I drove two people below in this way because the rest of us wanted the deck to ourselves." Many, however, are seasick on the darkest nights and in the seclusion of their staterooms with their eyes closed or bandaged, this having been suggested as a preventive measure, and the blind are not exempt nor are the near-sighted less subject to the malady than those of normal vision. Changing impressions on the organs of sight do not explain why negro slaves brought from Africa, prisoners and animals confined between decks, with no opportunity of seeing the water nor anything but the rigid beams and bulkheads of their quarters, are often so violently affected.

An ingenious theory of the phenomena of this affection attributes them to irregular variations of barometric pressure from the rising and falling of the waves, causing oscillations of the column of blood within the larger vessels, but these oscillations continue and are greatly exaggerated by heavy seas and gales, with very great barometric depression, as when the *Idaho* encountered the memorable typhoon of the 21st of September, 1869, during which the mercury fell to the unprecedented level of 27.62 inches, without a single case of seasickness, even though excessive fear and the feeling of impending death aided in depressing every one on board, some of them inexperienced recruits.

Roewer, an experienced German ship's surgeon, while not denying that the motion of the ship may cause a variation in the blood pressure in the large vessels, does not believe that this is sufficient to have any mechanical effect upon the contents of the cerebro-spinal cavities, but he attributes "the morbid train of symptoms collectively called seasickness to a confusion of the muscular sense," through which the individual has learned to maintain his relations with surrounding objects on land but is unable to do so under the unstable conditions prevailing on board a vessel floating in a yielding medium; and he asserts that as soon as this muscular sense is educated to the new environments of the body, the sickness disappears. This is controverted by the facts that among old seafarers, who never "lose their sea-legs," many are occasionally affected, and some even among officers in the naval and merchant services always so, and that the vertiginous feeling not uncommonly persists long after the return to the land, as in the case of a lady recently returned from Europe, who was not seasick on the outward passage but was so coming back, who told me a few days ago that although she had been at home a fortnight, she was still so dizzy she could scarcely walk upon the pavement.

Duval, Martialis, and others have a somewhat similar view of the causation of seasickness, holding it to be the result of the continuity of the muscular contractions necessary to maintain equilibrium. This is also Aronssohn's idea, though he gives to vertigo a principal rôle. Moussoir believes the confusion of "*le sens d'espace*" to be the fundamental fact in the *mal de mer.*

The majority of the writers on this affection have sought a mechanical explanation of its phenomena. Kéraudren, Andreux, Jobard, Bennet, Curie, and others declare the condition to be due to the movements which the motions of the vessel impress upon the abdominal viscera. Thus, Curie says that the oscillations of the vessel cause the intestines to press against the diaphragm and lift it and then to recede and allow it to fall, so that all one has to do to keep from being seasick is to learn how to hold one's breath at the proper time and thus make the diaphragm a resisting barrier to the intestinal impulses; but this theory does not account for the sickness which some suffer as soon as they put foot on the deck of a vessel securely moored in a protected port where there is scarcely a perceptible swell from the incoming tide, nor explain why the diaphragm should be so indifferent to the rough jolting which the abdominal contents receive during a ride on a hard-trotting horse.

Gilchrist found a satisfactory explanation in a similar shaking up and down of the brain in its bony case, caused by the irregular motions of the vessel, thus producing repeated slight concussions of the brain, which transmitted its impressions to the peripheral nerves.

Armand Jobert defined naupathia to be a sort of idiopathic indigestion, the peristaltic movements of the stomach struggling to resist those set up by the ship, and thus interfering with the normal physiological process. "Still the same idea," says Skinner of others who consider nausea marina and vomitus navigantium as all there is of the affection and who look beneath the epigastrium for their origin. "They wish to make the stomach guilty of causing seasickness. It may have accomplices so to speak, but still it is the most to blame. The stomach, nevertheless, has little to do with the production of naupathia."

Forget, in his "Médecine Navale," unites the views of Darwin and Kéraudren, and attributes the *mal de mer* to the bewildering mobility of surrounding objects, occasioning the vertigo, as well as to the unaccustomed agitation of the abdominal viscera, with this a greater or less nervous influence, producing the nausea and vomiting.

According to Jobard, while the essential cause is mechanical, it is none the less true that the ship's smells second its influence, and also that the malaise and vertigo which precede the nausea and vomiting are in part the result of the impression on the sense of sight of the shifting horizon, the rolling waves and the water rushing by the ship's side and in its wake.

Fonssagrives, holding that the symptoms of a severe attack of seasickness—the vertigo, tendency to syncope, sensory disturbance, vomiting, involuntary discharge of fæces and urine, etc., are analogous to those of slight concussion of the brain, elaborated the idea that the resultant of the two arcs of a circle described by the axis lines of the ship set up a centrifugal jarring of the brain, which with the faulty distribution or displacement of the sub-arachnoid fluid determined the cerebral commotion through the lack of the proper protecting bath of the brain, such a centrifugal tendency being likewise the cause of the vertigo and nausea experienced in the act of waltzing.

Autric made the shocks inflicted upon the brain and spinal marrow by the violent flux and reflux of the cerebro-spinal fluid induced by the movements of the vessel, responsible for the phenomena of seasickness, but these ordinarily disappear in a few days or hours and children uniformly escape them, although the alleged ebb and flow of the fluids upon the brain and spinal cord continue with unceasing activity.

Wollaston with greater plausibility ascribed the condition to a cerebral congestion, and Pellarin with no less confidence to cerebral asthenia, caused by a centrifugal force called into action within the vessels in consequence of the oscillations of the ship, the brain not receiving sufficient blood for its normal stimulation, analogous to what occurs to persons who have been bled and who toward the end of the act become nauseated, vomit and faint, a condition similar also to the nervous sedation induced by tobacco.

Pellarin does not deny that there may be, as M. Auzias-Tuxenne says, "by reason of the slowing of the general circulation a stasis of venous blood in the cerebral sinuses," which he holds takes place as in migraine, but says that it is the want of the excitation of the nerve centres by arterial blood which is to blame, instancing the sickening feeling that is experienced in fogs and drizzling rain, which cool the surface and thus retard the capillary circulation. Pellarin attributes the nausea of early pregnancy to the fact that the womb becomes the centre of blood afflux at the expense of the brain.

The fundamental fact in seasickness in Marshall Hall's opinion is a modification of the circulation of the spinal cord, an idea elaborated by John Chapman in his work on "Functional Diseases of the Stomach," which attracted considerable attention about thirty years ago on account of the peculiar therapeutic measures advocated by him for the prevention and treatment of the condition. These were based on his theoretical explanation of the cause of seasickness as proximately due to an abnormal amount of blood in the nerve centres along the back, and especially in those segments of the spinal cord related to the stomach and the muscles concerned in vomiting, this condition being induced by the movements of the vessel, he says, in three ways: first, through the brain; second, through the ligaments of the spinal cord; and third, through the abdominal and pelvic viscera. Fatal results are, however, exceedingly rare, and when they do occur an examination is impracticable, so that there has been no confirmation by post-mortem appearances of the assumed spinal hyperæmia.

An explanation of an entirely different character was that advocated by Semanas. Early in the century a physician of Lyons propounded a theory that seasickness, which he not inappropriately proposed to call "marine vertigo," was due to a marine intoxication. Semanas, reasoning from the supposed efficacy of quinine in preventing and relieving the headache and other symptoms of seasickness, assumed the existence of a sort of marine miasm, a toxic agent introduced into the economy, and consequently dependent more on the agitation of the surface of the water than on the movements of the vessel; but were this the case, the traveller on a canal-boat ought to be no less subject to the affection, and the gliding of a steady side-wheel steamship through a smooth sea would also release and diffuse the mischievous miasm.

Guien divides the causes of seasickness according as they act on the whole nervous system or on some of the nerves in particular of animal life, the most frequent being those that act on the nerves in general, through the rolling and pitching of the vessel, great cold affecting the cutaneous surface tending to the same result. Impressions on the senses of smell, hearing, and sight are special causes of cerebral vertigo, which reacts on the spinal cord, gastric nerves, and general sympathetic system.

Nature.

From this cursory *résumé* of the views of the prominent writers on seasickness, it is evident that there is substantial agreement among the most recent authorities as to the place of naupathia among the neuroses, and that they chiefly differ in giving especial importance to this or that point of departure from the condition of perfect health, according as they have convinced themselves of the efficacy of the particular form of preventive and remedial treatment each one advocates. Seasickness is primarily and essentially a marine vertigo. The rolling and especially the pitching of the vessel disturb the accustomed relation of the individual to surrounding objects, and cause, generally through the sensorium by any of its approaches, or through the agitation of the visceral contents of the great cavities, or the disturbance of the circulation, or through all these simultaneously, a succession of irregular and unexpected shocks to the nervous system, which fails to effect the necessary adjustments for equilibrium, inducing at first vertigo, "nervousness," malaise, and headache, and after a sometimes inappreciable interval, the sympathetic system being involved, nausea, vomiting, and prostration. While the onset of mild attacks may determine a temporary increase of blood in the cerebrum, it is certain that the lessened arterial tension due to the vasomotor disturbance later deprives the nerve centres of their proper stimulus, and the consequent anæmia of these centres results in weakness of the heart and dilatation of the vessels. This reciprocal depression of the circulation and function of innervation accounts for the continuance and progressive severity of the condition, ending in syncope, nervous prostration, and death, unless relief be found either through the cessation of the prime disturbing cause, by the smoothness of the sea and lessened movements of the vessel, or the habituation of the body to its variable surroundings, and the diminished sensitiveness of the sensorium to the impressions on the organs of the senses, or finally through medical interference by giving greater tone to the circulatory apparatus and increased activity to the centres of nervous influence.

Treatment.

So long as the surface of the ocean is tossed and turbulent, so long as human beings are so variously constituted that the scent of the rose, so sweet to the many, is nauseating to a few, and so long as men and women continue to do the insane and insanitary things they ought not to do, so long will the roll of the seasick continue. The advertised preventives and cures will benefit certain individuals, who by a little prudent forethought might have avoided their necessity, while others will continue to succumb despite all precaution or any kind of preliminary dosing. The experiment of the *Calais-Douvre*, the twin ferry-steamer constructed to transport passengers across the Straits of Dover between Dover and Calais, with the idea of preventing rolling, and the attempt to preserve the horizontal position under all conditions of sea and weather, and thus get rid of both pitching and rolling, by the Bessemer suspended saloon, and also the swinging state-rooms introduced in several steamships and yachts have proved failures. Dr. Neveu-Derotrie constructed a *fauteuil-de-mer* with the same object as that of the numerous patented swinging berths and bunks, which have been successively tried and abandoned, but none of these is superior to the ordinary man-of-war hospital cot or sailor's hammock, which does add greatly to the comfort of even old sea-goers, and especially when hung near the middle of the vessel, in a well-ventilated locality, where the water cannot be seen, the noise of the engine heard, nor the odor of grease and galley be smelt, does diminish to a minimum the chances of becoming seasick. If there had been any more reason to expect success from the various mechanical appliances to prevent the movements of the intestines and other viscera, in the shape of belts, bands, compressors, supporters, and cuirasses, than from the attempt to prevent the squirming of a bag of eels or earth-worms, it might have been expected that the female corset, laced until the pelvic organs protrude from the vagina and the diaphragm has absolutely no room to descend, would effectually protect its wearers; but in fact these furnish the greatest number of sufferers, while young girls and children, who never wear them, uniformly escape. While Kéraudren and others only sought to keep the entrails still by abdominal compression, Jobard advised the band around the abdomen less to fix the bowels than to determine the flow of blood to the head as in decubitus, but the seasick are really benefited by the removal of all constrictions, especially such as interfere with respiration. It has been proposed to subdue the rebellious diaphragm by inflating the lungs and holding the breath as long as possible, or to regulate the respirations by the movements of the vessel. It was hoped that the enormous size of the modern mammoth passenger steamship would have insured stability and lessened the liability to seasickness, but the hurricane and the cyclone toss it about as lightly as they do the sail-boat, yacht, and steam-tug, and by reason of the trembling of the vessel from its forcible impact against the wall of water and the vibration from its powerful engines, the ocean racer despite its luxurious equipment is more uncomfortable than the slower-going steamship, which does not hesitate to sacrifice time to stress of weather by heaving to or slowing down.

No less futile are the attempts to insure the body against the possibilities of seasickness by preparatory exercises with swings, rocking-horses (*l'équitation artificielle* proposed by Guiot), oscillating planks, gyratory chairs, and gymnastic evolutions designed to educate the senses to indifference to the disturbances of equilibrium, or to put them in such a condition of indifference by a preparatory course of medical treatment, or to find an antidote in any single medicinal agent or combination of drugs. Extravagant pretensions have been made in behalf of sodium bromide, bromo-chloral, codeine, cocaine, amyl nitrite, cerium oxalate, and other sedatives which are claimed to destroy the susceptibility of the nerve centres to the inciting causes of seasickness. Beard recommends potassium bromide as a prophylactic, urging its use several days before going on board, and pushing it to the physiological effect of bromism. According to Dutton, a combination of ammonium bromide with aromatic spirit of ammonia and chloroform, administered several days before embarking, by its sedative effect upon the brain and nervous system will certainly prevent the vaso-motor disturbance and the afflux of blood to the brain and spinal cord, but while some may be apparently benefited, there is no reason to hope this will be any more invariably effective than the preliminary exhibition of stimulants of the cardio-vascular and cerebro-spinal systems, such as champagne, strychnine, ammonia, and the aromatics, with the view of fortifying the system to resist attack. As a rule, however, it is wiser to keep drugs where Bacon wore his bag of saffron, over the stomach, rather than put them inside, and in most cases the simplest and perhaps as efficacious as any of the procedures advocated is to follow the counsel of Rey, as quoted by Irwin, "*fermez les yeux et prenez patience.*" I was once convinced that I had warded off an attack by applying to the epigastrium the pole of a sulphate of mercury battery, a very convenient little apparatus, which had been given to me for the purpose, but the same lady having made subsequent passages unscathed without its use, I am not sure that it had anything to do with her exemption at that time.

Nevertheless something may be done in the way of precaution. The ocean traveller usually prepares for a sea voyage as a bride is gotten ready for marriage, by weeks of anticipatory sur-excitement and bodily fatigue; and just as the young girl starts upon her bridal tour in a feverish asthenic condition demanding rest, quiet, and care for its reparation, and wholly unfitted for the performance of those new functions, which involve still greater excitement and expenditure of vital force, so does the housewife, exhausted by onerous domestic preparations for departure, after a long period of sewing, shopping, packing, and farewell visiting, involving reckless exposure, loss of sleep, and irregular meals, undertake to encounter physical environments that may be such as to demand the utmost constitutional vigor. It is little more surprising, therefore, that the offspring of the one should bear the marks of parental infirmity than that the other should become actually and seriously ill. Other imprudents, both men and women, who have been spared physical exhaustion, gorge themselves at the last moment with confectionery or disorder their digestion by injudicious indulgences, which increase their susceptibility to seasickness beyond the power of bromides, chloroform, or strychnine to overcome.

Manifestly all these are things to be shunned. The individual should be placed in the best possible condition before undertaking a sea voyage. Unnecessary fatigue on the eve of sailing should be avoided, the diet carefully regulated, and habitual constipation overcome. Ordinarily a dose or two of "liver pills" or saline cathartics are considered appropriate and sufficient preliminaries. Others invite seasickness under the belief that an attack is beneficial, based on the fact that improvement in health is the usual result of a voyage at sea, the improvement, however, occurring in spite of the vertigo, nausea and vomiting, and not in consequence of them. Myself an enthusiastic advocate of the incalculable benefits of ocean-therapy, I would not have it thought, as commonly believed, that seasickness has any desirable share in the good accomplished. Valetudinarians even more than mere pleasure-seekers should guard against it in every possible way. The gain in appetite, weight, and strength will be all the more rapid than if even a mild attack of seasickness supervene.

The invalid passenger apt to become seasick should lie down, immediately upon going on board, in a well-ventilated apartment, or on deck as near amidships as possible, out of the direct rays of the sun but well protected against chilling, with the eyes closed that the water may not be seen, nor the sweep of the masts, spars, and rigging across the sky, and away from the influence of the sounds and smell of the engine and galley. A moderate amount of easily digested food should be taken at short intervals, such as palatable nutritious soups, broths, or gruel, or even after nausea and vomiting may have occurred, matzoon, kumyss, or kumysgen, which have been found, in my own experience, the simplest and most satisfactory substitutes for other food. Guien, after some sensible advice not to allow the stomach to remain empty and racked by ineffectual retching, recommends the ingestion of mulled wine, a small glass of grog, curaçao, or eau de menthe, and advocates the use of a "*dragée orientale et aromatique*" of his own composition. In my experience, none of the alcoholics with the possible exception of very dry champagne, which is, however, in no way superior to kumysgen, are desirable in the early stage of seasickness, and equally objectionable are the few drops of creasote, ether, or chloroform, which are favorite remedies especially with English travellers. On one occasion, when I had prepared for an extended experimental use of matzoon, on a passage to Europe, I was disappointed that among all the crew and passengers, both steerage and cabin, there was but one case of severe seasickness, the slight discomfort of the rest disappearing on the second day, the weather having been delightful, the sea smooth and the air invigorating during the whole passage. The single case was a Dutch nun, one of a dozen sisters of a religious order returning to Holland, whose gastric disturbance was so great in consequence of previous inanition and enervation, that the matzoon was the only article she retained and of which she became so fond as to refuse all other food. Subsequently, I have used kumysgen and clam juice as dietetics in seasickness with great satisfaction, and in ordinary cases I believe them to be all that is necessary, with perhaps a large mustard-leaf over the epigastrium or brisk massaging with alcohol to give additional confidence and assurance of speedy recovery. When acid or effervescing drinks are craved, as lemonade, seltzer, champagne, or citrate of caffeine, they will be found beneficial in tiding over the period of apprenticeship to the sea. Sometimes coffee and tea are acceptable, but beer and alcoholic drinks generally only aggravate the nausea.

When medical interference does become necessary, and this is only in the class of serious cases, the particular treatment is usually influenced by the pathogenic views of the prescriber. Chapman depended upon the ice-bag to the spine with the object of lessening the amount of blood in the nerve centres along the back, but aside from the inconvenience of application and the discomfort from unavoidable wetting of the bedding and clothing, it is of doubtful utility, as much good probably resulting from the ancillary remedies recommended—draughts of ice-cold water, pieces of broken ice held in the mouth, warmth to the abdomen and feet. Semanas administered quinine to neutralize the toxic poison he believed to be derived from the marine atmosphere, and a formidable list of other remedies to meet special indications has been advertised in the periodicals; this includes amyl nitrite, ammonia, antipyrin, atropine, belladonna, the bromides, cannabis indica, chloral, chloralamid, chlorodyne, chloroform, ether, nitroglycerin, paraldehyde, phenacetin, sulphonal, etc. Of these, the potassium bromide recommended by Beard as a prophylactic, and ammonium bromide in combination with aromatic spirit of ammonia and spirit of chloroform recommended by Dutton for the same purpose, as already stated, are also urged by them respectively as effective remedial agents. Charteris claims that equal parts of potassium bromide and chloralamid, in the combination known as "chlorobrom" is a palatable, potent, and safe remedy, which, through its chloralamid ingredient, "secures sleep by its action on the cerebral cortex and has but slight influence on other parts of the nervous system of voluntary life. It has no direct action on the heart and circulation. It does not reduce arterial pressure, and its administration is not attended with any disturbance of digestion or depression." The effervescing hydrobromate of caffeine is a favorite remedy with Danvers, and an elixir of caffeine and cocaine has been used by Skinner and found acceptable by patients. The latter claims to have also had good results from the hypodermatic administration of caffeine, in thirty centigram doses, after the formula of Tanret and Dujardin-Beaumetz, to wit: Caffeinæ, 4 grm.; sodii salicylatis 3 grm.; aquæ destillatæ, q. s. ad 10 cc., each cubic centimetre of which contains forty centigrams of caffeine. The treatment in which he has especial confidence, however, is the subcutaneous injection of one gram of a solution of two centigrams of atropiæ sulphas, and four centigrams of strychniæ sulphas in forty grams of peppermint water, the dose being half a milligram of the atropine salt and one milligram of the strychnine every two hours until three doses shall have been taken, euphoria usually being produced after the first or second. The indication sought to be met is to overcome the diminished arterial pressure caused by paresis of the centres of innervation of the heart and arteries by stimulating these centres and the unstriated muscular fibres. In severe cases when the vomiting persists and prostration is impending, I have no doubt this treatment will be useful, the hypodermic form of administration being especially commendable under such circumstances. Cases of this gravity should be placed in swinging hospital cots in well-ventilated apartments; bottles of hot water should be kept to the feet and along the limbs, which should be briskly rubbed with alcohol; pledgets soaked with the same should be placed the whole length of the spine and over the thorax and abdomen; and alcohol should also be sprayed abundantly over the clothing and bedding until the atmosphere of the apartment shall be saturated with its vapor, the inhalation of which I have found one of the most effective calmative and soporific agents in the irritability and insomnia of anæmia and nervous prostration from whatever cause, when alcohol taken into the stomach is objectionable. When kumyss or clam juice cannot be ingested, rectal alimentation must be practised to prevent inanition. Patients at this stage, which, fortunately, has never come within my experience, and cases complicated by concurrent affections, require such treatment as the exigency may suggest.

Persons dangerously affected by seasickness after one experience ought not to repeat it, especially those already invalid, who are probably the only ones who suffer critically. Manifestly those affected with acute maladies, serious diseases of the circulatory apparatus, especially aneurism, hæmoptysis, mental disorders, and diseases of the spinal cord should not undertake a sea voyage. Epileptics are apt to have paroxysms of the disease developed, and while asthmatics occasionally find relief, they are as often made worse. The menstrual function is rarely affected, as before stated. Abortion is seldom induced in the enceinte, while the vomiting of early pregnancy is often averted, and the excessive bleeding attending fibroids and other uterine complaints is materially lessened, as an incident of the general improvement.

THERAPEUTIC USES OF A SEA VOYAGE.

As I have said elsewhere in advocating the therapeutic advantages of ocean climate, the invalid seeker after health may hope to gain by a sea voyage, especially a long one on board a comfortable sailing-vessel, if season, course, and destination be judiciously chosen, or by prolonged residence on some ocean island where all the conditions of sea life prevail, first of all *rest*—of mind and body—a condition of absolute insouciance, and relief from the cares and distractions, the daily worries and anxieties of life, the interruptions and noise and turmoil and excitements which railroads, telegraphs, and newspapers bring into the very sick-room on land. If not so completely bedridden (in which case he ought not to go to sea at all) but that he can lie in an easy-chair on deck, he will be able to breathe and bathe in an air that is barren of every impurity and with every inspiration experience a sense of pleasurable invigoration. If seasickness is not an indomitable idiosyncrasy, he will after a few days, when he "gets his sea-legs," as sailors term it, find inexpressible delight in pacing the narrow bounds of the quarter-deck, or hurricane-deck, and lengthen the hours of this gentle exercise until they become whole watches long. Once accustomed to the motion of the vessel and of the sea, nausea will give place to appetite, which is sharpened as well for old salts as landlubbers, and the plainest food will be taken with unwonted relish betokening improved assimilation. If care be had to overcome the usual tendency to constipation, ingestion may go on without restraint. The eagerly anticipated meal hours become the eventful marks of the passage of time. The whilom patient eats and sleeps and wakes to find new zest in the simple employments of the day. Rocked in the cradle of the deep, the ocean's lullaby soothes the sufferer into forgetfulness of his ills.

A long sea voyage in temperate climates on board a clean roomy vessel, well outfitted and equipped, is a judicious therapeusis for chronic invalids of almost every class; but to be effective the ship itself must be free from the dirt, dampness, and bad ventilation which under aggravated circumstances made scurvy, ship fever, and dysentery actual scourges of sea life, and in a less conspicuous degree show their effects in the rheumatism, phthisis, and diarrhœa which are needlessly frequent among sailors. The slowly convalescent from whatever disease—from protracted fever, chronic diarrhœa, nervous astheny—the incipient pulmonary case, the feeble, fretful valetudinarian, the overworked business man, the broken-down woman, the child with slender hold on life, if seasickness be controllable, will, after a few days of pleasant sailing, recover appetite, and often recuperate with marvellous rapidity. The inebriate removed from the temptation or the means of self-gratification, the not inconsiderable class of cases of sexual exhaustion and of other forms of impaired nervous power; the thousands of victims of their own follies, who fill the sanatoria, private hospitals, and health resorts; the irritable and melancholy hypochondriac and misanthrope are especially benefited by the quiet restfulness of a long sea voyage in pleasant weather, when the assimilative process becomes the dominant function, and the worn brain and flaccid vessels and feeble lungs are beguiled into feeding on what will make them strong again.

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Besides the leading writers on seasickness, of whose works use has been made in the preparation of this article, and of whom a list is appended, the literature of this affection comprises a considerable number of English, French, and German theses and several hundred articles in medical periodicals, the latter chiefly advocating certain special methods of treatment. The authors quoted or referred to are the following:

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MOUNTAIN SICKNESS.

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MOUNTAIN SICKNESS.

The atmosphere which surrounds our earth exerts upon its inhabitants a definite influence that can be demonstrated in two directions. The one, mechanical in its nature, is based upon the weight of the atmosphere. Let us imagine a column of air extending to the extreme limit of the atmosphere; then the pressure of the upper layers condenses the air in every deeper layer so that a tension is produced which is greater in proportion to the proximity of the layer to the ground. At the sea level the pressure of the air balances in the barometer a column of mercury 760 mm. (29.922 inches) in height, and the tension of the air equals this pressure. If we calculate the pressure which it exerts upon a definite space we find that every square centimetre at the surface of the earth bears a weight of 1,030 gm., or every square inch a weight of about 15 pounds. We do not feel this pressure because it is not unilateral, for in every part of the body the pressure from one side is opposed by an equal pressure from all other sides so that no solid or liquid particle can be displaced. In the upper layers the air pressure diminishes in proportion to the height of the barometer; at an altitude of about 5,800 metres it amounts to 380 mm. and hence is only half as great as at the sea level.

In proportion to the varied tension and density of the air an alteration of its pressure affects mechanically our respiratory activity.

In the other direction the density of the air, by reason of the oxygen it contains, affects the conditions of our vital activity. The absorption of oxygen through the lungs into the blood is to some extent influenced by the tension of the oxygen; it takes place more rapidly in denser air than in rarefied air in which the tension of the oxygen is decreased in proportion to the rarefaction.

It is true, populations living at an altitude between 3,000 and 5,000 metres are not affected by the rarefaction of the air of these elevations, for their lungs and their respiratory activity are adapted from the time of their birth to the thinner air. But the inhabitants of lower regions frequently suffer from the altered conditions in the beginning of their sojourn at a greater height, and the symptoms manifested by them are grouped under the name of "mountain sickness." The symptoms of mountain sickness are most pronounced when great elevations are ascended without preparation and they are felt chiefly while the person is in motion and much less when he is at rest. Experienced mountain climbers are rarely attacked and then only in the highest altitudes; of late, therefore, since mountain climbing has become a popular form of exercise, we hear much less than formerly of the ill effects of mountain sickness.

The mean altitude at which mountain sickness generally occurs in Europe may be assumed to be 3,000 metres (9,840 feet), with an air pressure of 530 mm. Many persons are affected at a lower elevation and Saussure mentions a case at an altitude of 1,600 metres (5,248 feet). The writer, too, knew a lady who was forced to leave Davos, at an altitude of 1,600 metres, because she respired with difficulty.

Saussure states that he was not affected until he reached a height of 3,700 metres. Others may go still higher before they are attacked; for instance, the von Schlagintweit brothers, who felt the symptoms in the Himalayas first at an altitude of 5,000 metres (15,240 feet) and then but slightly.

How greatly frequent repetition of mountain climbing facilitates respiration in rarefied air may be learned from the well-known traveller Güssfeldt, who, when ascending Aconcagua in Chile, which is nearly 7,000 metres high, experienced dyspnœa only when beyond half the ordinary atmospheric pressure at an altitude of about 6,000 metres, and even this did not prevent him from reaching the summit.

Symptoms.

The changes caused by mountain sickness affect the respiration and the action of the heart, also the activity of the muscular and nervous systems so far as it depends immediately upon the amount of oxygen contained in the blood, and lastly the distribution of blood in the vascular system. In describing the symptoms as they occur at successive altitudes I shall follow the reports of ascents of Mont Blanc which correspond in the main with all other reports from different localities. The height of Mont Blanc is 4,800 metres (15,786 feet), barometric pressure at the summit 430 mm.

Attentive observers have found that the pulse beat is somewhat accelerated when the body is at rest, even at the slighter elevations of about 1,000 metres. In like manner, in ascending a higher mountain, it is possible to recognize the effect of the diminished atmospheric pressure below the height at which symptoms generally occur, by the fact that under exertion the frequency of the pulse increases more markedly than during the same activity at a lesser height.

The first symptoms manifest themselves by the respiration becoming less ample so that frequent pauses are necessary in order that deeper breaths may be taken. On continuing the ascent, an unusual weakness is felt in the legs which soon give out, so that after every few steps one is forced to stop or to sit down. On endeavoring to overcome this, the movements of the heart, already accelerated, are painfully felt; great palpitation of the heart and throbbing of the arteries, dyspnœa, flickering before the eyes, and the sensation of an approaching faintness enforce rest. After a very brief interval the feeling of strength returns, and the walking is resumed, to be soon followed by the same weakness.

On continuing the ascent a sensation of slight nausea is soon superadded, at times associated with vertigo; eating is impossible and even the stronger alcoholic liquors are rejected. This is accompanied sometimes by an irresistible drowsiness, confusion, and embarrassed mental activity. The individual becomes despondent, and indifferent about anything that may happen, even dangers. In milder cases only lassitude, nausea, or headache is felt.

The further increase in the symptoms consists in a distinct swelling of the veins of the face, arms, and hands, lividity of the face, sometimes injection of the eyes, severe headache, coldness of the hands and feet, nausea increased to vomiting. Some persons are affected with an exudation of blood from the overfilled capillaries of the mucous membranes of the eyelids, the nose, the gums, and the bronchi.

During rest, especially in the recumbent position, all the symptoms abate; during the descent they are much less marked and soon disappear under the rising atmospheric pressure.

When an eminence at which a person was first affected is visited again after a short interval the symptoms usually occur in a milder degree and continued practice finally confers immunity. During the first ascent of Mont Blanc by Saussure all the guides were affected, and the like is reported for some length of time of the succeeding ascents. But since in recent years the ascent has been frequently repeated we seldom hear that a guide has suffered in any marked degree from mountain sickness. Hemorrhages, too, have not been reported of late from the Alps. Saussure has stated that mules suffered on the higher Alps; travellers in the South American Andes frequently report that the animals -horses, mules, oxen, and dogs—succumbed to mountain sickness unless they had been born at the elevation or had been previously acclimated there. Beasts of burden often perish when goaded and when their pack is not lightened; well-nourished horses suffer particularly and readily bleed from the nose. Cats seem to be acclimated with much greater difficulty than other domestic animals. In Asia camels are also affected.

The impression gained from the narratives of those who have ascended Mont Blanc is strengthened in some directions by the reports of mountain sickness in the South American Andes where great heights are reached on the backs of mules and hence the traveller is more rarely and less markedly affected on the march, because he himself is not in active motion. After arrival, however, the symptoms occur, and Pöppig[1] has described his sensations in the first few days of his sojourn in the mountain city of Cerro de Pasco, Peru, 4,400 metres (14,432 feet) above sea level. He says: "When the newcomer first goes out in the morning he is surprised by an inexplicable lassitude, painful dyspnœa, slight headache, and a sensation of a rush of blood to the head and lungs. In vain he tries, by the greatest expansion of the chest, to fill the lungs with the vital element; it seems as if he were in a vacuum. The steep lanes become troublesome, the feet are scarcely able to carry the body, the knees bend, he is forced to rest often. Finally the will is paralyzed, the mind becomes dull and dejected." He also makes mention of the accelerated pulse without fever, the nausea, feelings of faintness, the sensation of cold in hands and feet, and the inclination to sleep but without finding rest. At night, he says, the oppression is greater; reading is impossible because the eyes are sensitive.

After a longer sojourn the symptoms disappear. In from six to twelve days, says Pöppig, the worst manifestations are gone, but lassitude and difficult respiration remain for a long time. During the first year no European can use his powers in his customary manner or as at lower altitudes, but gradually he becomes acclimated.

According to Tschudi,[2] the inhabitants of low countries, when once accustomed to breathe in the rarefied air, can easily ascend even high mountains and move about as freely as on the coast.

I shall report some special observations upon several of the above-mentioned symptoms in order to demarcate the conditions and their occurrence more sharply.

The frequency of the pulse was first counted by Saussure[3] on August 4th, 1784, in his own person and in two others at the summit of Mont Blanc (barometer 435 mm.), after four hours of rest. Before the ascent the pulse of the three persons was 49, 60, and 72 respectively; at the summit it was 98, 112, and 100 respectively. During a subsequent ascent of the Roche St. Michel[3a] on Mont Cenis, barometer 499 mm., Saussure noticed that the pulse had become more frequent in persons who still felt ill after two hours of rest than in those who had remained unaffected.

Lortet,[4] a physician and physiologist of Lyons, observed the frequency of the pulse on the march during his first ascent of Mont Blanc on August 16th and 17th, 1869. His pulse, which was 67 in Lyons, was 80 on the march at an altitude of 1,500 metres (4,920 feet); at 3,000 metres (9,840 feet) where he passed the night, it was 116. Until then Lortet had felt no inconvenience. The next day at 3,460 metres (11,349 feet) the pulse rose to 128, at 4,500 metres (14,760 feet) to 136, and finally on the summit of Mont Blanc under the full effect of the mountain sickness to 160 and more, when it fell again after two hours' rest to 90–108.

These observations were subsequently confirmed by Dr. Calberla[5] during the ascent of Monte Rosa—4,695 metres (15,379 feet)—though to a less extent, since no symptoms of mountain sickness appeared either in him or in his two guides. Moreover, there are exceptional cases in which neither accelerated pulse nor dyspnœa was observed on the summit of Mont Blanc. Thus a companion of Dr. Piachaud[6] could finish the last stretch to the summit on a run and his pulse was no quicker than before the ascent, while Piachaud himself felt miserable and sick.

Lortet also noticed that the pulse curve at the greater altitudes became dicrotic—a symptom which was likewise observed by Lazarus and Schirmunski[7] in the pneumatic chamber—under the effect of rarefied air, with other symptoms of mountain sickness.

The respiration was studied by Lortet by means of an apparatus, the anapnograph, which showed the depth of the respiratory acts. At an altitude of 3,050 metres (10,000 feet) breathing was not yet impeded; at rest he counted 24 respirations which, however, were less deep than at a lower elevation. At 3,900 metres (12,800 feet) the respirations had begun to become shallower, the frequency had risen to 36, and shortly after reaching the summit respiration, even on standing still, was short and restricted; there was a sensation as if a hoop had been drawn around the chest. At the summit the symptoms gradually abated, after two hours the frequency had again become 25, but the slightest movement induced dyspnœa. During his second ascent of Mont Blanc, ten days later, Lortet felt much less inconvenience.

In this connection I must not omit mention of a noteworthy observation by Lortet with reference to the temperature of the body during the march in the ascent. He measured the temperature by means of specially constructed small maximal thermometers which could be placed under the tongue. These showed even at an altitude of 1,500 metres (4,920 feet) during the march a lowering of the temperature which rapidly regained its normal height whenever the marcher stood still. The difference increased with the altitude and near the summit amounted to rather more than 3° C. (about 5.4° F.). The temperature of the air did not fall below $-3°$ C. (26° F.). A similar observation had been previously made by Dr. Marcet,[k] who also discovered that with the onset of nausea the body temperature fell. Later observers were unable to confirm the lowering of the temperature during the march, but it should be noted that none of them suffered from mountain sickness, to which Lortet and Marcet were subject. In P. Bert's work [21] we find observations on animals, which demonstrate that the lowering of the body temperature is one of the symptoms of mountain sickness.

The best description of the failure of muscular power is furnished by Saussure.[3b] According to him, at a distance of about 300 metres from the summit of Mont Blanc the grade is only 28° or 29° and the tract is quite free from danger. But in the rarefied air the strength is rapidly exhausted. Even when near the goal he was obliged to sit down after every fifteen to sixteen steps in order to breathe and rest. He says: "This kind of fatigue is absolutely irresistible. When at its height, the greatest danger would not enable us to take another step. . . . When I attempted to force myself my legs gave out; I felt the approach of a fainting fit associated with violent palpitations and a beating of the arteries in the head. My eyes grew dim, quite apart from the glare of the light, since I wore a double veil which completely protected the face." In another place he says furthermore: "On the other hand—and this is the second peculiarity of this kind of fatigue—one feels a complete restoration of the strength from the mere cessation of motion. Even without sitting down, after two or three minutes the strength seems to have returned so completely that one thinks one's self able to reach the summit with a single effort."

At the summit Saussure felt well, barring a slight sensation of nausea, so long as he kept quiet. But as soon as he directed his attention intensely, even for a moment, to his observations, he had to rest two or three minutes to enable him to breathe. During the descent he felt no inconvenience because, as he says, in going down a mountain the action of the diaphragm is not interfered with.

I shall add another observation of Saussure's with reference to the drowsiness which sometimes attacks mountain climbers at the greater elevations. He says (§ 559): "After a few moments' rest all feeling of the preceding fatigue seems to have completely disappeared. Nevertheless we see in a very little while that every one who had no special occupation has fallen asleep, in spite of wind, cold, or sunshine, and often in very awkward positions. It is true, fatigue induces sleep also in the plains, but never with such rapidity, especially after every trace of fatigue, as upon mountains, seems to have disappeared as a result of the brief rest."

Noteworthy, too, are other observations made with reference to the conditions during the night's rest. A. Vogt,[9] who was free from mountain sickness, noticed when he camped in the open air at night on the Aaresattel in Switzerland at an altitude of 3,000 metres that he and the guide lying beside him respired twice as often as in the lowlands. In others the nocturnal accelerated respiration is associated with inconvenience. Thus Moorcroft[10] on different occasions in the Himalayas, at an altitude of 5,000 metres, whenever he was about to fall asleep was awakened by dyspnœa and a sensation of anxiety. At such times some deep respirations relieved him. A similar report is made by Mrs. Hervey[11] from Thibet at an altitude of about 5,000 metres. Often when in bed at night and on the point of falling asleep she was awakened by dyspnœa and violent palpitation. She was forced to sit up in bed in order to be able to breathe. Others have made a like report.

Some authors state that they found it difficult to speak, without giving further particulars. A fuller statement is made by Humboldt,[12] who reports that new-comers in Quito (3,000 metres—9,840 feet) experience impeded respiration, particularly when talking rapidly.

Owing to the altered distribution of blood which induces a predominating fulness of the veins and capillaries with a relative depletion of the arteries, loud speech favors the occurrence of hemorrhages, because it suddenly retards the circulation through the lungs. Boussingault[13] in the Peruvian highlands strictly forbade his companions loud speech or calling. He had observed on the Antisana that one of his servants had suffered a hemorrhage in consequence of calling loudly to a companion who had strayed from the path. A similar case is related by Pöppig: an Englishman of his acquaintance in Cerro de Pasco who played on the flute had to give it up because every attempt produced bloody expectoration. Mrs. Hervey, too, states several times that the nightly occurrence of great oppression with dyspnœa was associated with bloody expectoration, which was increased when she spoke.

This very pronounced tendency to hemorrhages, like the other inconveniences, persists only at the beginning of a sojourn. During the first days, says Pöppig, shaving should be omitted because the skin, particularly if light-complexioned and delicate with a tendency to become chapped, bleeds readily.

I may add that Tschudi[2] mentions, besides bloody expectoration, also a bloody diarrhœa as a frequent concomitant symptom of the *veta* or mountain sickness in the Peruvian highlands.

A decided influence upon the increase of the symptoms seems to be exerted by climatic conditions, among which I include the stronger reflection of light and heat, especially from snow-covered surfaces, and the greater dryness of the air.

The reflection of the light, acting upon the retina, causes headache, sometimes nausea. A similar effect can be produced by reflection from the bare ground or from the surface of the water in the lowlands, but on mountains and snow-covered surfaces it acts more energetically. For this reason veils or goggles are worn as soon as the snow line is reached. On Mont Blanc and the Andes the combined influences of the reflection of light and heat sometimes excite dangerous inflammations of the eyes, called surumpe in the Andes; the skin, too, where acted upon, swells, becomes painful and chapped, and later on desquamates.

In elevated regions the air is less moist than in the lowlands, for according to Hann one-half of all the atmospheric aqueous vapor is contained in the inferior layer to a height of 2,000 metres. In the Andes particularly the dryness is so great that in the Peruvian plateau the flesh of the smaller food animals, dried in the air, can be preserved for future use. The dryness favors the occurrence of the above-mentioned cutaneous inflammation, of fissuring of the skin and lips, and is the cause of the frequently increased thirst. In moist air and rainy weather the suffering is said to be less, which is partly owing probably to the decreased radiation when the sky is overcast.

The influence exerted by the temperature as such upon mountain sickness varies according to whether the body is at complete rest or is excited by the effort of climbing. Cooling of the body at rest often provokes the symptoms and H. von Schlagintweit[14] relates that during a prolonged sojourn at an altitude of more than 5,000 metres on the plateau of Karakorum, where the two brothers made observations for some time and took little physical exercise, the afternoon or evening wind regularly robbed them completely of their appetite. Those sleeping in tents at night awoke with a sense of oppression when the breeze sprang up. A similar observation is reported by Dr. Marcet. One night on Mont Fréty, 2,200 metres (7,200 feet), he awoke during the great cold of the morning with a sense of nausea which, according to a written communication, he recognized from experience as one of the symptoms of mountain sickness.

It is said that in the tropical regions of the Himalayas and the Andes mountain sickness attacks travellers at a greater height than in Europe. In those regions much greater altitudes are attained on horseback or mules; hence the traveller is much less exposed to the affection than in European mountains, where the same heights must be reached on foot. Moreover, in India especially, several day's marches are necessary before the greatest altitude is attained, so that a gradual transition takes place. Not rarely we read among the statements of travellers that at a certain height they experienced the onset symptoms of mountain sickness, but on the following days, at a greater elevation, they remained free from it.

According to the reports of the French academicians who about the end of the last century visited the South American Andes for the purpose of measuring the first degree of the meridian, and the statements of von Humboldt, mountain sickness occurs in the valley of Quito at an altitude as low as about 2,500 metres (8,200 feet).

Windless localities, whether warm or cold, usually give rise to greater inconveniences, as for instance the Corridor on Mont Blanc at an altitude of about 3,900 metres (12,800 feet). On similar principles we might explain the observation that in mining regions of the Andes symptoms usually occur in certain parts of the way which are well known to the mule drivers. The natives ascribe the fact to metallic exhalations and therefore call the disease sorroché (antimony) and veta (metallic vein).

These influences, which in the first place suppress evaporation from the body that on such occasions is often perspiring freely, weaken the nervous force. But the nervous system is concerned in other ways in the production of the symptoms. Great fatigue and exhaustion by hunger increase the symptoms; for this reason we find the advice given, despite the aversion to food, to take a very little nourishment frequently, whereby the condition is improved as in seasickness. This probably explains also the custom in the Himalayas and in Afghanistan of using stimulating and refreshing articles, such as certain fruits and onions, during the ascent. On the other hand, the prospect of an early attainment of the goal, exhilaration, and mental expectancy, enable one to overcome the attack more readily, in the same way as on shipboard the officer can perform his duties in spite of the seasickness, while the passenger succumbs to the affection without resistance.

The bodily condition of different persons is not without importance with reference to the power of resistance toward the disease. The old are acclimated with more difficulty than the young; from the Andes it is reported that stout and plethoric persons with much fat are attacked sooner and suffer more, and this is true also of animals, especially horses. Anæmia does not seem to favor the occurrence of the disease. The well-known geologist Burmeister,[15] who intended to ascend the mountains from Argentina, was attacked on the foot hills by a prolonged intermittent fever. Though hardly fit to travel he traversed a mountain pass at an altitude of 4,548 metres and was surprised that he remained free from the trouble in spite of his weakness. Once only he had a transitory feeling of giddiness lasting but a minute.

The natives of such altitudes remain free from all inconveniences. The traveller d'Orbigny [16] relates that on the way to La Paz in Bolivia, while crossing a pass at a height of 4,500 metres, he, with his driver, horse, and dog, suffered greatly from mountain sickness. He was hardly able to breathe, had severe headache, palpitation, nausea, and epistaxis. He then describes the impression made upon him when he saw two native messengers climb and pass the rocks at a rapid pace and with the greatest facility.

The symptoms thus far reported may be separated into two groups. Under the influence of an unaccustomed respiratory activity phenomena occur which indicate on the one hand a marked lack of oxygen in the blood and on the other hand an alteration in the distribution of the blood. Among the former belong the loss of muscular power in the legs, the nausea, the lowering of the bodily temperature, etc.; among the latter, the overfilling of the veins and the capillary system, together with the acceleration of the pulse as a sign of the corresponding decrease of arterial fulness. The drowsiness and the headache may be caused or favored both by arterial depletion and lack of oxygen and by the impeded efflux from the veins of the head.

History.

The attempts to explain these remarkable observations have occupied the scientists and physicians of Europe for two hundred years. A glance at the history of the various views regarding the cause of mountain sickness shows the changes they underwent with the progress of scientific investigations.

The first statement in the literature is found in the book on South America by the Jesuit J. Acosta[17] who had crossed the Andes in 1590. He ascribed the cause to the subtle and more penetrating air in the passes of the Andes. It was not until half a century later that the existence of the pressure of the air was demonstrated and the barometer and the air pump were invented, so that scientists could begin to observe the effect of a vacuum and of rarefied air upon living organisms. One of the earliest authors, Robert Boyle,[18] says in 1670 that the tension of the air, lessened in accordance with the altitude, must have caused the difficult respiration in Acosta. Later experiments showed him the escape of air from the blood under the air pump, from which he reasoned that when the air pressure is greatly reduced the air contained in the blood distends the vessels and might cause circulatory disturbances. A few years later Guide[19] found that the lungs of animals dying in rarefied air (in vacuo) became atelectatic and sank in water, and this observation was confirmed about fifty years later by Muschenbroek.[20] Others, it is true, have denied this, but P. Bert[21] has recently, under similar conditions, likewise found in the lungs atelectatic portions of greater or less extent.

Albrecht von Haller[22] in his great work summarized the observations made up to his time. He says: "In rarefied air the expansion of the lungs is rendered difficult because the concomitant action of the tension of the air has decreased and the heart therefore receives less blood and acts less energetically, whence comes the general loss of strength. The external pressure upon the blood-vessels which enabled them to resist the force of the heart is diminished, and they rupture more easily. When the air pressure is much reduced the danger becomes greater because the air contained in the blood expands."

Haller could take account only of the mechanical effect of the air pressure upon respiration, for at that time oxygen was not known as a vital element. The cause assigned by Haller to the swelling of the veins and the hemorrhages was later briefly designated as "turgor toward the exterior" which became effective under decreased air pressure. The rupture of the vessels presupposes their greater repletion such as may be observed in the superficial veins in mountain sickness, and according to the statements given this evidently extends to the entire venous and capillary systems. Therefore the explanation given by Haller fails to account for the augmented volume in one half of the vascular system which would necessitate a diminution of the contents in the other half. For it is not conceivable that a decrease of the air pressure, affecting all the vessels equally, could produce so extensive a displacement of the contents of the vascular system, and hence something else must be superadded in order to explain the altered distribution of blood.

As regards the liberation of air from the blood, Hoppe's[23] observations in 1857 have shown that air is not given off from the blood until the pressure has sunk to 57 mm. which would correspond to an elevation of more than 20,000 metres (65,600 feet).

After Lavoisier's labors in 1876 had demonstrated the importance of oxygen to life, Saussure[24] (1787) was able to explain teleologically the frequency of respiration which increased with the altitude, by the necessity for counteracting the dilution of oxygen by rapid breathing. He also connects the accelerated circulation with the frequent respiration, but assumes that the lowered pressure upon the blood-vessels likewise contributes toward rendering the circulation more rapid.

With reference to the degree of dilution of the oxygen contained in the inspired air with which accelerated respiration occurs under normal air pressure, observations have been made upon dogs which are as sensitive as men to a lowered pressure. According to these, increased frequency of respiration does not begin until the amount of oxygen in the air is far below that corresponding to the air pressure of 435 mm. on Mont Blanc. Hence here, too, some additional cause must be active.

A long time after Saussure the attempts to explain mountain sickness moved in the same circle, some of the various associated conditions being emphasized as the chief contributing causes; for instance, the dazzling by the snow, the greater effect of heat radiation, and the dryness of the air; furthermore, the bodily fatigue, the exertion during hunger, the peculiar quality of the air of certain localities such as sultriness, exhalations from the ground, etc.; finally, the greater cooling of the body by evaporation and radiation. The contributory effect of the air pressure in retaining the femur in its socket, discovered by W. and E. Weber in 1836, was long cited in explanation of the fatigue in the legs at great altitudes. This explanation, however, had to be abandoned when it became evident, according to the measurements and calculations made by Dr. Farabœuf[21a] by request of P. Bert, that the air presssure must fall to 190 mm. before the joint could become loose. This would correspond to an elevation of about 12,000 metres (39,360 feet). The consideration of the mechanical effect of the air pressure on respiration was meantime lost sight of until Pravaz[24] (1850) again recurred to it. He pointed out that the lungs are expanded only by the air pressure, which being diminished would overcome with difficulty the elastic resistance of the lungs. His only follower was Campana[25] (1870).

So long as it was believed that oxygen was taken up by the blood by physical absorption in the same way as it is by water, most writers agreed that the increased respiratory activity is caused by the lessened amount of oxygen, but that it does not suffice for completely compensating the deficiency in the blood. In this way some of the symptoms were explained.

As early as 1837 Magnus had found that the air pump withdraws from the blood a much greater quantity of oxygen than the amount which should be present in accordance with the law of physical absorption. In 1857 it was demonstrated by Lothar Mayer and Fernet that the oxygen is chemically attracted and bound by the blood cells and it was believed that the assumption was justified that the blood could saturate itself with oxygen under any accessible air pressure. Therefore another explanation of the symptoms of mountain sickness had to be sought for.

In 1861 Jourdanet,[26] who had passed considerable time on the plateau of Mexico, called attention to the necessarily slower diffusion of oxygen from rarefied air and cited this fact as the cause of a relative lack of oxygen. In this way he explained the anæmic conditions which he found generally prevalent under the atmospheric pressure of the Mexican plateau, 585 mm. (2,000 metres—6,560 feet). As to the distribution of blood he adhered to Haller's hypothesis. Although in view of the health and strength of the inhabitants of places lying at much greater altitudes than Mexico anæmia cannot be recognized as a general condition, still Jourdanet must be given the credit of having first called attention to a possible cause of a diminished amount of oxygen in the blood.

In 1866 Gavarret,[27] who had preceded Fernet in asserting that the absorption of oxygen by the blood must rest not on a physical but on a chemical attraction, advanced a new hypothesis which is related to the mechanical theory of heat. He assumed that the augmented exertion required by the ascent of high mountains demands the consumption of an increased amount of heat, that therefore a larger quantity of carbonic acid is formed, whose accumulation in the blood produces the symptoms of mountain sickness.

He was followed in 1870 by Lortet,[4] whose observations are given above, but who accords the first place in the causation to the lowering of the bodily temperature, and puts the increased production of carbonic acid in the second place.

An accumulation of carbonic acid in consequence of great exertion cannot be the cause of mountain sickness because the same symptoms occur also under the diminished air pressure in the pneumatic chamber where the body performs no active labor, and the same is true of balloon ascensions. The lowering of the bodily temperature, however, was observed by P. Bert[21 b] in the pneumatic chamber as a concomitant symptom of mountain sickness in animals. Accordingly the fall of temperature must be looked upon as one of the symptoms and cannot be their cause at the same time.

In the seventh decade of this century P. Bert[21] published some important papers which gave a fresh impetus to the question. He had examined the blood from a number of dogs, which had been taken from the arteries under the influence of reduced atmospheric pressure. He invariably found a diminished amount of oxygen as compared with the normal quantity in the blood of the same dog. The deviations from the normal, under equally reduced pressure, were very irregular in different dogs, sometimes considerable, sometimes unimportant. He then made experiments regarding the absorption of oxygen with blood agitated with oxygen under the ordinary and under a diminished atmospheric pressure. At the ordinary temperature he found no difference in the saturation, but when the agitation was performed at the temperature of the blood, from 38° to 40° C. (100° to 104° F.), there was a slight decrease in the contained oxygen when the atmospheric presssure was reduced one-half. Bert concluded from this result that under reduced atmospheric pressure at the temperature of the blood there occurred a dissociation of the combination of oxygen with the hæmoglobin so that the blood could not be completely saturated.

The difference, however, was but slight and therefore he believed that acclimation to a life in rarefied air may occur, the person abstaining from an often excessive consumption of oxygen and managing to get along with less. He compares the consumption of oxygen with that of food which in many people is likewise larger than is required for the needs of the body, while others possess the same endurance with less nutrition.

Bert's experiments were soon repeated by others with improved methods and his results were confirmed, though in a minor degree. Thus Setschenow,[28] of St. Petersburg, demonstrated in 1880 that when the atmospheric pressure was reduced to about one-third of the normal, corresponding to an altitude of 8,800 to 9,000 metres (28,800 to 29,500 feet), the variations in the chemical absorption of oxygen by the blood were quite insignificant, even at the temperature of the body. Soon after A. Fränkel and I. Geppert,[29] of Berlin, reached the conclusion that the blood of living dogs at rest was saturated normally with oxygen at a reduction of the air pressure down to 410 mm., corresponding to an altitude of rather more than 5,000 metres (16,400 feet). At one-half of the atmospheric pressure, 380 mm., they found on the average a slight diminution in the proportion of oxygen in the blood, but this was not so great as to be beyond compensation by an augmented respiratory activity.

Finally in 1891 Bohr,[30] of Christiania, by experimenting with dogs found that as a rule the tension of the oxygen in the blood was greater than in the air of the lungs. This would be a proof that the absorption of oxygen by the blood is less dependent upon the tension of the oxygen in the atmosphere than one might be inclined to believe. On the other hand we know that under increased astmospheric pressure oxygen is absorbed more rapidly by the blood than under the ordinary pressure,[31] and as regards reduced pressure Setschenow[28] found that the quantities of blood agitated by him with oxygen required a longer time for their saturation under a diminished tension of oxygen than under a greater pressure. Hence we are justified in assuming that when the air is greatly diluted the oxygen is more slowly diffused in the blood than when the air is denser, but not so slowly that it could not be easily compensated for by an appropriate respiratory activity. In a recent communication Dr. A. Löwy[32] says that a person experimented on in the pneumatic chamber under a pressure of 410 mm. was able to overcome the oppression and work on the ergostat for half an hour without inconvenience, although the absorption of oxygen during such work was four times the normal.

An Attempt to Explain the Symptoms of Mountain Sickness.

There is no doubt that with increasing rarefaction of the air a limit must be reached beyond which the lungs cannot absorb enough oxygen for the maintenance of life. This, however, is not the question with which we have to deal here, searching rather for an explanation of the symptoms which begin to occur far below this limit.

The fact that a number of persons remain free from mountain sickness and that the rest can sooner or later adapt themselves to the conditions of respiration in rarefied air proves that a normal absorption of oxygen is possible up to a height of 5,000 metres and beyond, which is often reached in the summer by herdsmen. Therefore it is not the dilution of the oxygen in the first place which produces mountain sickness. We must search for another cause which under certain circumstances would be apt to prevent the saturation of the blood with oxygen in the rarefied air, the possibility of which is recognized. This cause must at the same time include the reason for the altered respiration and distribution of blood. The latter, however, has been quite neglected by modern authors, the majority of whom are satisfied with the theory advanced by Haller.

If we bear in mind that mountain sickness, under equal external conditions, will sometimes attack and sometimes spare the same person, we must look for the cause which produces it in the individual only. The cause must lie in the quality of our organs in so far as their activity is subject to the influence of the air pressure, besides being acted upon by exertion and the nervous system. Exertion and lessening of nerve force take a prominent part in the intensity of the symptoms.

The only organ of the human body whose activity can be altered by the effect of atmospheric pressure is the lung. During inspiration the activity of the respiratory muscles is assisted by atmospheric pressure in expanding the lungs and overcoming their elastic tension. The return to the contracted state during expiration is effected, as a rule, without the aid of muscular power; it is the result of the inherent elasticity of the lungs. Expiration, therefore, is a purely mechanical process which must be influenced by an alteration in the density of the surrounding air.

The result of the change in the activity of the respiratory organs thus induced is clearly shown by the overfilling of the venous system which can be produced only by a more contracted state of the lungs; for it is well known that in an expanded state of the lungs the larger veins are visibly depleted, while the collapse of the organs causes a stagnation of the venous blood.

In the first place I wish to call attention to the fact that even under ordinary atmospheric pressure we meet with cases in which laborious ascent with a more contracted state of the lungs produces not only alterations in the distribution of the blood, but also other symptoms resembling those of mountain sickness, though they are less in degree.

When a person unaccustomed to climbing, rapidly ascends a steep declivity covered with soft snow, frequently sliding back, it often happens that after every few steps the knees give out, vertigo occurs, there is dazzling before the eyes and a sensation of impending nausea. The heart palpitates violently, the face becomes livid, and the veins swell. A moment's rest with deep breathing suffices to dispel these symptoms. A more contracted state of the lungs ensues here because during continuous and laborious exertion of this kind the abdomen remains retracted and the diaphragm cannot descend. The incomplete expansion of the lungs prevents the entrance into them of a certain amount of blood which accumulates in the veins. The absorption of oxygen then is somewhat restricted by the incomplete respiration and is still further reduced by the distribution of blood, for an excess of blood in the veins cannot contribute to the absorption of oxygen. The fatiguing exercise, however, would on the contrary necessitate a greater supply of oxygen, and its absence in combination with the slighter filling of the arteries causes the transitory inconveniences.

The symptoms just described correspond exactly with the similar symptoms experienced at high altitudes, the only difference being that the latter occur sometimes even when the person is at rest or is making but slight exertion.

At great elevations, therefore, a contraction from the mean state of the lungs must be possible even when the body is at rest, the cause thereof residing in the diminished atmospheric pressure, there being a *relative increase of the elastic force* of the lungs as compared with the diminished density of the surrounding air.

Let us first consider the effect of diminished density upon *expiration.* We have seen that this is a mechanical process so long as it occurs in the normal way without the aid of muscles, and, like every escape of air, is affected by the rise and fall of the density of the surrounding atmosphere.

Special experiments have shown that air expelled by pressure from minute openings escapes more rapidly when the density of the surrounding atmosphere is diminished and more slowly when it is increased. The rapidity of the escape follows a definite law; it is in inverse proportion to the square root of the air pressure.[33]

In accordance with this law, under the reduced pressure on Mont Blanc at an altitude of 4,800 metres (15,750 feet), expiration would be completed about one-fifth more quickly than under ordinary pressure.

Under diminished atmospheric pressure the contraction of the lungs is not only more rapid but it is also more intense. Both of these facts I have been able to demonstrate by comparing my observations under increased and ordinary atmospheric pressure with those of Mermod[34] made under ordinary pressure at Strasburg and with diminished pressure at an altitude of 1,100 metres (3,600 feet).[35]

The accelerated expiration under diminished atmospheric pressure will produce first a more rapid and shorter breathing; this, well known from the reports of laymen and physicians, has been proved also during rest by careful observations.

By means of a gas-meter Mosso[36] in September, 1882, compared the respiration of a man who was not affected with mountain sickness on the pass of Theodul at an altitude of 3,333 metres, or 10,932 feet (barometer 510 mm.), with the respiration of the same man in Turin and found that the average frequency of respirations was increased on the pass from 11 to 14, while the volume of the single respirations sank from 508 to 459 c.c. Very similar results were obtained by myself in 1890 by the same method with a person in the pneumatic chamber under a pressure of 515 mm.[37]

Hitherto it has been assumed that the acceleration and shortening of the respirations are caused by the decrease of oxygen in the rarefied air. The objection to this theory, as has been pointed out above, is that no acceleration of respiration is observed when, under the ordinary pressure, the oxygen in the respired air is diminished in proportion to the altitude, within the limit of 9,000 metres. Some experiments made with dogs seem to indicate even that a decrease of oxygen in the air causes at first deeper respirations with lessened frequency.[21]

Inspiration under diminished atmospheric pressure is not accelerated as is expiration, but on the contrary it is somewhat retarded. By the greater contraction of the lungs during expiration the mean diameter of the smallest bronchioles is lessened for the succeeding inspiration; and this, as experiments have shown, is bound to retard the expansion of the lungs somewhat, especially at the beginning of inspiration, since less air can enter through smaller openings during an equal time.[38]

The sensation of facilitated respiration experienced after ascending a slight eminence rests rather upon the contrast between the labored breathing during the ascent and the relief after arrival than upon any real acceleration of inspiration.

A further influence of the relative rise of the elastic tension of the pulmonary tissue upon respiration will naturally be appreciated only with deeper breathing because at the beginning of the respiration this tension is still quite weak. In the beginning it does not exceed that of a column of mercury four or five millimetres in height and becomes greater only in the course of the expansion of the lungs so that at the extreme dilatation of the thorax it may reach 30 mm.

Therefore, while even at a great elevation respirations of slight depth can be performed with facility, difficulty will be experienced only during deep breathing. This becomes clearly evident by the observation made by numerous experimenters that the vital capacity of the lungs or their expansibility as measured by the spirometer sinks more or less markedly under diminished atmospheric pressure. The lungs can no longer be as freely dilated as under normal atmospheric pressure.

In making this measurement the expanded position of the thorax must be maintained for a few moments. In the rarefied atmosphere this maintenance is rendered difficult because the resistance which is opposed by the denser atmosphere to the contraction of the lungs has lessened.

In my own person, without suffering from mountain sickness, I observed under an atmospheric pressure of 515 mm. a decrease of the capacity of my lungs from 3,950 c.c. to 3,700 c.c., that is, of 250 c.c.; in another gentleman the decrease was from 3,680 to 3,510 c.c. The repeated efforts I made to expand the lungs left a sense of pain under the sternum which continued for a number of hours.[38]

At a pressure of 515 mm. when respiration is not yet rendered difficult the absorption of oxygen by the blood is normal. But when with marked diminution of the atmospheric pressure symptoms of mountain sickness occur the capacity may sink still farther because the respiratory muscles do not obtain sufficient oxygen. This was what happened in the case of P. Bert.[24] His normal capacity, amounting to 3,750 c.c., diminished in the pneumatic chamber under a pressure of 430 mm. to 2,560 c.c., that is, by 1,190 c.c., about one-third. At the same time under the effect of the rarefied air he suffered greatly from vertigo, nausea, muscular tremor, and embarrassed mental power. The inspiration of an air mixture rich in oxygen which he had prepared enabled him, however, to bear subsequently a much greater reduction of pressure.

P. Bert was more sensitive to the effects of atmospheric pressure than most other persons—a fact which points to a greater elasticity of his lungs.

Both the retarded inspiration and the more rapid expiration are also noted by v. Vivenot[39] in describing his observations in the pneumatic chamber under the atmospheric pressure of Mont Blanc (435 mm.). First there occurred a dyspnœa-like acceleration of breathing, soon followed by a need for deep inspirations. He found inspiration impeded and expiration facilitated.

Quite noteworthy are the respiratory curves obtained by Lortet[4] with the anapnograph during his ascent of Mont Blanc. They showed an embarrassment of inspiration, rising with the altitude, owing to a diminished amplitude of the respirations and to an increasing difficulty in maintaining a uniformly progressive expansion of the lungs. The expiratory curves, on the other hand, showed even at the height of 3,050 metres, or 10,000 feet (Grands Mulets), that at the close of the natural contraction of the lungs the abdominal muscles were called into play in order to prolong the expiration and increase the change of air. At this altitude he had not yet felt any symptoms of mountain sickness. Abdominal respiration, however, became markedly prominent, under the effect of mountain sickness at the summit, in the straight horizontal lines suddenly interrupted which are traced by the expired air. Under normal atmospheric pressure, on the other hand, the expiratory line shows a gradual decline corresponding to the lessening rapidity of the contraction.

The above-described alterations in the course of inspiration and expiration are obviously liable to produce a more contracted state of the lungs, for they enforce the contraction of the lungs while embarrassing their expansion. This leads to the overfilling of the venous system, to the hemorrhages, and finally to the atelectasis of smaller or larger portions of the lungs observed by P. Bert and others in animals dying under increased rarefaction of the air. As long as the heart pulsates in such a case the more contracted lungs are overcharged with blood, as they are found at the autopsy. When the atmospheric pressure which keeps the pulmonary vesicles expanded has fallen to the minimum it may easily happen that the capillaries of a vesicle are so overfilled with blood as to expel the air from it. Such a process, however, must cause a stasis in the circulation of the surrounding parts and thus give rise to an atelectasis of an entire lobule. All observers agree that the atelectatic portions can be again inflated.

It is easily recognized that a relatively increased pulmonary elasticity eventually produces the same result, as regards expansion of the lungs, as does a diminished atmospheric pressure, to which latter influence the earlier authors ascribed the embarrassed respiration. It is only another name for the same thing, but it has the advantage of an experimental basis.

We cannot escape the influence of the reduced density of the air or resist it because we do not become conscious of the change in the atmosphere. How true this is is shown by the observation of Bert[21] who, whenever in his experiments the atmospheric pressure had fallen to about 500 mm., was no longer able to whistle. While he had adjusted the muscles as usual for whistling, yet he could make no sound because he was no longer master of the state of the lungs and the current of air.

The same cause explains the breathlessness produced by speaking observed by Humboldt[12] in Quito, since in uttering a sound more than the customary amount of air is lost by the rapid contraction of the lungs.

Under the ordinary atmospheric pressure any demand made on the respiratory activity is readily responded to because the respiratory movements can at once obey the stimulus of the requirement; but under diminished pressure this is not so easily possible, for our respiration is adapted by habit to a denser atmosphere in which expiration proceeds more slowly and the regulation of the respiratory movements as a rule is automatic and beyond our consciousness of an effort. It is particularly for this reason that we find it impossible to make allowance for a change in the density of the air the occurrence of which is unperceived by us.

When under the usual atmospheric pressure the need of a fresh inspiration occurs before the lungs have attained their greatest contraction—and this is probably most frequently the case—we exert our respiratory muscles at the right moment and with the necessary force. Under diminished atmospheric pressure, however, the contraction of the lungs by that time has always advanced farther than it would have done under the usual pressure and hence the effort is retarded and the exertion of the muscular force which is gauged in the customary manner no longer corresponds to the task. The result of the respiration is unsatisfactory, it is rapidly repeated, and other symptoms may follow.

This is best appreciated in the breathing of one who is on the point of falling asleep, at which time the regulation of the respiration is exclusively under the control of the habit acquired under a greater atmospheric pressure. The frequency of respiration is then much increased and is often followed by oppression and dyspnœa which are connected with a more contracted state of the lungs. The affected person is forced to sit up and ease himself by some deep inspirations.

When, during active exercise, such a tendency is subjected to influences which reduce the nervous force, then a person will often no longer be able to overcome the embarrassment of the more and more labored respiration and a point may be reached where sufficient respiration will become altogether impossible.

This explains why a weakening of nervous force by fatigue, hunger, a sultry or a cold atmosphere, or discouragement will contribute to the aggravation of the conditions. If the inconveniences were felt before but slightly or not at all, such influences may develop them into a more severe attack, since the regulation of the respiratory activity is lost.

This will sometimes happen even after complete acclimation seems to have been acquired. A striking example of this kind is furnished by the description given by Tschudi[2] of an attack of mountain sickness suffered by him after he had for an entire year traversed altitudes of 4,000 to 5,000 metres in Peru, both on mule-back and on foot, without any inconvenience. One day at an altitude of 4,500 metres he had been riding upward the whole morning without breakfast and had lost his way. The mule was tired, he had to dismount and leading the animal by the rein find his way while climbing in the sun. His exertions became violent through excitement and suddenly he began to feel the influence of the diminished atmospheric pressure by losing control of his respiration. I give his description in his own words: "With every step I was seized with a previously unknown discomfort, I had to stand still in order to get air, but I barely succeeded; I attempted to walk, but an indescribable anxiety overcame me, my heart beat audibly against the ribs, breathing was short and abrupt, a world weighed upon my chest. My lips became tumid and fissured, the small capillary vessels of the eyelids ruptured, and the blood exuded drop by drop. My senses failed, my vision became clouded, and trembling I was forced to lie down on the ground."

After half an hour's rest in a semi-conscious condition he was able to remount his mule and continue his way.

The attack in this case was brought on by the unusual exertion; then Tschudi, owing to the weakening of his nervous force, lost control over his respiration which previously seemed to be firmly established. His condition resembled that of a stammerer who, apparently cured, again begins to stutter in a moment of embarrassment, his futile efforts only adding to the trouble.

Tschudi's description shows that the dyspnœa increases greatly during the attack—a fact which may be learned also from the reports of Pöppig and Lortet (*vide supra*). This symptom finds its explanation in an observation by v. Basch,[40] according to which overfilling of the pulmonary capillaries produces a condition called by him rigidity of the lungs. In this state the lungs can be only imperfectly expanded even under normal atmospheric pressure and with the most vigorous respiratory efforts. Under reduced pressure it becomes still more difficult and the increasing failure of the efforts to get air is incomprehensible to the patient. The result is an indescribable confusion during which ineffectual efforts, as in Tschudi's case, are repeated to the point of exhaustion until finally the enforced rest again establishes compensation in the circulation and restores the balance between the supply and the consumption of oxygen.

It is a matter of general experience that physical exertion may provoke the symptoms of mountain sickness in persons heretofore exempt. A rider may continue well as long as he remains in the saddle; when he dismounts and walks he is attacked. At a meeting of the Geographical Society of Munich Dr. Essenwein, a surgeon in the German navy, spoke of a trip he had made on the mountain railway from Callao to Cerro de Pasco in Peru. At an altitude between 3,000 and 4,000 metres while in the car he was still perfectly well, but after he had got out, on the way to the hotel, he was attacked by great dyspnœa with violent palpitation; he felt as if he were about to be seized with pneumonia and was forced to return by the next train down.

Exertion causes a more rapid passage of blood from the arterial into the venous system and therefore larger quantities of blood are brought to the right heart. These would accumulate even under ordinary atmospheric pressure were it not that normally during an easy walk deeper inspirations are made, which permit of the reception and passage of larger quantities of blood through the lungs. As under diminished atmospheric pressure the lungs contract more strongly, this escape is cut off. The blood accumulated in larger amounts in the veins is debarred from the exchange of gases so that even at rest the limbs receive less oxygen. The demand for oxgyen, however, is increased during exertion; for according to the calculation of Katzenstein,[41] man requires during moderate muscular activity from three to four times, and during violent muscular exertion from five to six times, the quantity of oxygen needed in repose. It is clear that in air which is deficient in oxygen, like that of high altitudes, exertion is certain to increase the symptoms induced by defective respiratory activity.

The symptoms of mountain sickness are always associated with an acceleration of the pulse and this occurs even earlier than the sensations of inconvenience. The cause of it lies in the more contracted state of the lungs which acts in two ways upon the circulation. On the one hand it diminishes the so-called negative pressure in the pleural space and thereby reduces the resistance to the current of blood in the arterial system;[42] on the other hand it induces a less complete filling of the arterial system. Both of these conditions, however, contribute to facilitate the evacuation of the left ventricle and according to Marey[43] the pulse frequency increases in proportion to the facility with which the left ventricle can be evacuated. A noteworthy circumstance in mountain sickness is the occurrence of an accentuation of the heart beat of which the patients complain. An equally marked accentuation was observed by me in a case of a different nature in which less blood reached the left ventricle because in an early stage of pneumothorax a large part of the right lung was suddenly excluded from the circulation. It seems therefore than an accentuation of the heart beat occurs when the right ventricle is overfilled with blood, while the left ventricle and the arterial system contain less blood than in the normal condition.

In animals dying under diminished atmospheric pressure Hoppe[23] always found the right ventricle and the lungs greatly distended with blood, and the left ventricle empty.

The dicrotic quality of the pulse tracings which was ascribed to the influence of the lessened atmospheric pressure occurs under such circumstances only when dyspnœa is present; it is, therefore, a symptom of mountain sickness which may result both from the diminished resistance in the arteries and from an accentuation of the heart beat.[41] In persons who are not attacked the pulse tracing is unchanged.

In the pneumatic cabinet I have observed that the pulse tracings were normal when the atmospheric pressure was diminished one-half, provided respiration was free. But as soon as the respiration was restricted they became dicrotic and sometimes also showed signs of an accentuated heart beat. In such a case the pulse was always more markedly accelerated. It has been thought that the cause of the accelerated pulse[44a] should be sought in the lack of oxygen, but this does not hold good, for P. Bert[21c] has observed that in dogs subjected under ordinary atmospheric pressure to a gradual diminution of oxygen in the respired air there was from the beginning a lessened frequency both of the pulse and of the respiration until the oxygen in the air was reduced to about one-third of the normal amount. This would correspond to an altitude of about 9,000 metres, while the accelerated pulse occurs at an elevation as low as 1,000 metres; therefore it could not be due to the lack of oxygen any more than the accelerated respiration.

Under diminished atmospheric pressure P. Bert found that the rise in the frequency of the pulse always went hand in hand with the increase in the frequency of respiration. When the latter became temporarily slower the pulse likewise lessened in frequency. This indicates the close connection of the pulse rate with the state of the lungs; for with the more frequent breathing under diminished atmospheric pressure the respirations as a rule become shorter and more shallow, while the state of the lungs is more contracted.

In *balloon ascents* the symptoms are essentially the same as on high mountains, but they become perceptible only at a greater elevation because the laborious physical exertion is lacking, and their further course is likewise influenced by this fact.

For the first observations regarding the pulse we are indebted to a balloon ascent by Gay Lussac and Biot[45] on August 9th, 1804. At an elevation of 2,622 metres (8,600 feet) they noted an acceleration of the pulse in Gay Lussac from 62 to 80, in Biot from 79 to 111. During a second ascent on August 29th Gay Lussac found at an elevation of 7,106 metres (23,317 feet) that his respiration was markedly impeded and his pulse greatly accelerated. Glaisher,[16] the English investigator, during his ascent on July 17th, 1861, at an elevation of 5,500 metres (18,040 feet) first observed violent palpitation with acceleration of the pulse and impeded respiration; at a somewhat greater height his hands and lips became livid, a certain sign of a lack of oxygen in the blood; and at an elevation of 7,000 metres (22,970 feet) great dyspnœa set in, and he felt sick and wretched.

The greatest height reached by Glaisher was during an ascent on September 5th, 1862, when he lost consciousness at an elevation of

8,200 metres (26,916 feet). He describes the sequence of the symptoms which formed the transition to this condition. At first he could no longer move his arms and legs, then sight was lost, he could still hear his companion but was not able to answer. Finally he lost consciousness which did not return until the balloon in descending again reached an elevation of 8,200 metres. Withal he was free from anxiety or disagreeable sensations of any kind. Glaisher states in a later publication[47] that he had gradually accustomed himself to breathe in rarefied air so that in subsequent ascents he no longer observed the livid discoloration of the face and hands at an elevation of 6,000 metres (19,680 feet).

The altered distribution of the blood is not mentioned by aeronauts who have had no physiological training; but that it is present is shown by the result of the autopsy of the aeronauts Croze, Spinelli, and Sivel. They succumbed in 1875 to the influence of the diminished atmospheric pressure which had fallen to 266 mm. at an elevation of about 8,600 metres (28,228 feet). According to the report of the surviving Tissandier the mouth and nose of the bodies were filled with exuded blood.[21]

Although the aeronauts, by advice of P. Bert, had provided themselves with bags filled with air rich in oxygen, from which they refreshed themselves from time to time, still they grew so weak at last that they could no longer make use of them.

SUMMARY.

Briefly summarizing the results of this treatise, I think I may claim that the cause which produces the symptoms of mountain sickness in dwellers of the lowlands when they ascend to altitudes between 3,000 and 5,000 metres, and sometimes at lesser heights, is to be sought neither in the dilution of the oxygen of the air alone nor in the lessened atmospheric pressure alone. A third factor is superadded in the elastic tension of the lung tissue which becomes relatively greater under diminished atmospheric pressure. This increased tension induces changes in the mode of respiration and a more contracted state of the lungs. These again act upon the circulation and cause an overfilling of the venous system which at times gives rise to hemorrhages from the mucous membranes. The combined effect of these alterations is a temporary inability to supply the blood under all conditions with a sufficient amount of oxygen from the rarefied atmosphere.

The reason why persons who are at first attacked become gradually accustomed to the lessened atmospheric pressure is obvious: a new habit is formed, the force of circumstances teaching the individual unconsciously to adapt the muscular exertion required for the act of respiration to the altered conditions until the respiratory movements have become spontaneous and free from effort.

The natives of high altitudes seem to possess a bodily structure which is better adapted for respiration in rarefied air than is that of those born in the lowlands. The French traveller d'Aubigny[16] observed that the average height of the South American tribes, beginning with Tierra del Fuego, decreases gradually with the rising altitude of the mountain regions inhabited by them, and that the Quichuas, who live in the highest parts above 2,500 metres (8,200 feet), possess the lowest stature, averaging 1.60 metres (5 ft. 3 in.); but they are distinguished by massive shoulders, an arched chest, and a comparatively greater development of the trunk. The latter is longer, while the legs are shorter than in kindred tribes and very muscular. At an autopsy in the hospital at La Paz in Bolivia he admired the extraordinary size of the lungs. These statements are confirmed by Dr. Archibald Smith[48] for the region of Cerro de Pasco in Peru, and according to a verbal communication by H. v. Schlagintweit the build of the inhabtaints of the altitude of Thibet in the Asiatic plateau is similar, in contrast with the dwellers in the lowlands of India who are taller on the average. The smaller tribes of the Indian lowlands, as for instance of Bengal, possess the normal proportion of the trunk to the legs.

Acclimation.

There is no doubt, however, that a healthy and vigorous man, though born in the lowlands, learns to adapt his respiratory activity to the rarefied air after a shorter or longer sojourn in the highlands. The foremost requisite is a slower and deeper inspiration suited to the new conditions, for it can be demonstrated[49] that fewer and more vigorous respirations are more favorable to the absorption of oxygen than quick and shallow breathing. The greater rapidity of expiration at great elevations furnishes a gain in time which can be utilized for deepening the inspirations without increasing their frequency. But this can be done more easily during repose than during walking. While marching and ascending progress should be slow enough so that the blood which passes in larger amounts into the veins may have time to be transported through the lungs, lest it accumulate. The habit of such a mode of respiration, which can be acquired by repeated mountain ascents and by sojourn at higher altitudes, gradually strengthens the respiratory muscles and through practice gives us the capacity even unconsciously to apply the right degree of force and rapidity in expanding the thorax during respiration. Contributing to the evacuation of the lungs by retraction of the abdominal muscles, which Lortet unconsciously called into play, is likewise useful in supplying a larger quantity of oxygen.

Thus far it is not yet certain whether a discovery made by Viault[50] in 1889 at Lima has any influence upon acclimation; namely, that the number of blood corpuscles is found considerably increased after a very short time, in men who have changed from a lower to a higher elevation. Since then the fact has been confirmed even for slight elevations and it has been ascertained that the increase in number is associated with a diminution in size. Future investigations will have to furnish us further information as to the importance of this change.

A difference in the elastic tension of the lungs will render acclimation more difficult in some and easier in others. An instance of a greater elasticity was furnished by P. Bert, while Piachaud's companion, who was still able to run on the summit of Mont Blanc without showing any acceleration of the pulse, undoubtedly possessed large lungs of very slight elasticity. An essential condition for overcoming more readily the early difficulty of respiration is a sufficient mobility of the thorax, and it is for this reason that persons who have led a sedentary life suffer more severely than those who have been accustomed to physical activity. That plethoric, well-nourished persons succumb more readily and are more apt to suffer from hemorrhages may be due to the fact that when such men exert themselves a comparatively larger quantity of blood passes into the veins and hence the overfilling of the venous system may reach a higher degree. Fat persons, who likewise suffer more severely, usually have small lungs.

On the other hand, lean persons, like Burmeister, are said to be less subject to the disease and to recover more easily, even when they are anæmic.

A visit to very high altitudes is especially injurious to persons affected with heart disease and pulmonary emphysema.

Diagnosis.

The diagnosis of mountain sickness is easily made when the history shows a recent change of residence from a lower to a higher altitude, and the development of the symptoms is borne in mind. It may be mistaken for febrile states, just as Dr. Essenwein was in doubt as to his own condition. In such a case the bodily temperature will dispel the doubt, for mountain sickness as such is not associated with fever. When bloody expectoration occurs, which would be met with only at very great elevations, the concomitant symptoms and the examination of the thoracic organs will serve for the differentiation.

Prognosis.

The prognosis of mountain sickness as such is favorable, aside from the consequences occasionally resulting in the shape of inflammations of the eyes and skin, for these are caused chiefly by the radiation and by the dryness of the air at high altitudes. Attacks of syncope are on record; a few fatal cases from hemorrhage have been reported from hearsay evidence, but not one from personal observation.

Treatment.

The treatment of mountain sickness may be in part deduced from what has been stated above. As regards the *prophylaxis*, an ascent leading into the region where mountain sickness is apt to occur should always be preceded by an ascent of lesser heights with prolonged sojourn there, in order that a correct mode of walking may be practised and to adapt the respiration as much as possible in advance to the reduced pressure. Besides, in way of preparation, a series of sittings under increased atmospheric pressure in the pneumatic chamber will doubtless be of service, as it augments the capacity of the lungs.

For the ascent proper experienced mountain climbers advise that some nourishment should be carried along, small quantities of which may be frequently taken in order to avoid weakening of the nervous system by prolonged fasting, without overloading the stomach. In the Himalaya and adjoining mountain ranges they use in some regions onions, in others the refreshment consists in acidulous dried apricots and prunes; in the Andes the smelling of garlic is thought to be sufficient. In preparing for an ascent Tschudi recommends the drinking of an infusion of coca leaves which are in general use as a nervine in the Andes. Against the radiation of light and heat protective glasses and veils are to be worn as soon as a certain elevation has been reached. During periods of rest the body should be warmly muffled. During the march at high altitudes there must be no loud talking or calling, and respiration must be performed with closed mouth so as to avoid the drying of the palate and throat which often occurs to a painful degree. For the same reason it is desirable to be provided with a small bottle of water or cold tea for use in case of necessity, as the thirst is often troublesome. The attempt to quench the thirst with snow makes it worse. Only a small minority can bear spirituous liquors; coffee, tea, or meat-extract bouillon is far preferable at meal time.

When, after arrival at an elevated locality, a person suffers from mountain sickness it is best for him to remain for a few days at rest in a moderately heated room. The sensation of nausea is always lessened or removed by assuming the recumbent position. For the excitement of the circulatory organs they use in the Andes lemonade, ice, and gentle laxatives such as cream of tartar with tamarind pulp. When the rush of blood to the head and lungs is great Archibald Smith recommends the abstraction of blood, but this would be advisable only in very rare cases.

When the affection continues to be troublesome, the remedy in Cerro de Pasco is to send the patient to a village situated about three miles below and let him stay there a few days. On his return the symptoms have either disappeared or at least moderated.

According to Tschudi, in the Andes they rub garlic or the pounded fruit of capsicum to the nose of mules, horses, and other animals in the belief that it eases their condition. The chief reliance, however, is placed on bleeding the animals which not rarely suffer from intestinal hemorrhages. This is done by cutting off the tip of their tail or a piece from the ears, or by making an incision into the palate, or finally, which he thinks best, by slitting open the nostrils.

Aeronauts, when they reach elevations at which weakness begins to be perceptible, inspire a mixture of air and oxygen which they carry along in appropriate bags, in accordance with P. Bert's recommendation.

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OSTEOMALACIA.

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OSTEOMALACIA.

Osteomalacia is a disease characterized by softening of the bones. The softened bones give way under the weight of the body and the action of the muscles, and various deformities are produced in consequence. The disease is closely related to various forms of atrophy of bone. Very similar deformities may be the result of rickets. The distinction ordinarily made between rickets and osteomalacia is that rickets is due to an imperfect deposition of lime salts during the formation of bone; the bones contain the same amount of organic material but remain soft owing to the absence of the inorganic constituents. In osteomalacia, on the other hand, the bones become soft from the removal of the inorganic substances previously deposited.

In normal development bone is formed from osteoid tissue. Young osteoid tissue contains large numbers of cells and is formed either as a secretion product of these cells or by their metamorphosis into the tissue. Many of these cells disappear in the process, others become included in the forming tissue, and become the future bone corpuscles. Inorganic substance is deposited in the tissue between the cells. The growing bone is always surrounded by a layer of these cells. At the same time that the formation of bone is taking place there is always some absorption and removal of the formed bone. In the stage of formation the bones do not correspond with the shape of the adult bones. The architecture is constantly changing. In various places new formation of bone is taking place, and in others the formed bone is being removed. There is no transformation of cartilage into bone. The intercellular substance of the cartilage may become calcified and represent a provisional bone, but this is absorbed and its place is supplied by true bone formed from osteogenic tissue. The cells surrounding the growing bone are called osteoblasts. The absorption of bone is due to the action of large multinucleated protoplasmic masses, the osteoclasts. The edge of the bone where absorption is taking place is rough and indented, the osteoclasts lying in pits which they have excavated in the bone. In most pathological processes the new formation and atrophy of bone takes place in the same way as in the development of bone.

In osteomalacia all parts of the skeleton may be affected. Certain parts are usually more affected than others, and the disease usually attacks first the bones of the pelvis. The affected bones may be so soft that they can easily be cut with a knife. In some cases the disease seems to attack principally the interior of the bone. The shape of the bone may be little altered. A soft porous material is found within, and this is surrounded by a layer of comparatively dense bone. This condition is more common in senile atrophy of bone than in true osteomalacia.

In most cases, in addition to the various deformities of the bone produced by bending and partial fractures, there are other alterations. The bones may be very much thicker, the thickening in some cases affecting the entire bone, in others appearing as tumor-like masses along its course. In some places the bone structure has disappeared entirely, and a soft, grayish mucoid-like mass takes its place, or in other places there is a tissue of the same general character as connective tissue. In this tissue in an advanced degree of the process there may be larger and smaller cysts filled with a clear fluid, or the fluid may be viscid and brownish from an admixture with blood pigment. These cysts are surrounded by a firm layer of connective tissue. The color of the interior of the bone varies according to the acuteness of the process. When the process is most acute the marrow is intensely hyperæmic and shows numerous hemorrhages scattered through it. When the process is less active there may be a diminished degree of hyperæmia. In the typical puerperal osteomalacia the bones usually become affected in a definite order. As a rule the disease first appears in the bones of the pelvis and the alterations are more marked here than elsewhere. After the pelvis, in the order of frequency, the vertebræ, the ribs, and the sternum become affected. The lower extremities are more frequently affected than the upper. The disease may be limited to certain of the bones of the body or it may attack them all; but in all cases, although the most marked changes will be found in certain bones, all of the bones of the body will be found on closer examination to be more or less affected.

Deformities.

There are always various deformities produced by the disease. The softened bones will be not bear the burden of the body, and the deformities which result are due to the pressure of the body on the various bones. They may also give way under the action of the muscles, and bends and twists corresponding to the traction exerted by certain muscles are found. In all cases where the disease is at all advanced the bones will be found bent in various ways and showing numerous fractures. At the place where the angles found in the bone would appear to denote a simple bending of the bone there will be found more or less extensive fractures which have been partially healed by the formation of an imperfect callus. The presence of such imperfect callus masses contributes greatly to the deformity of the bone.

In the lower extremities there is always a bending of the bones outward. As a rule in the femur the greatest deformity is found in the angle of the bone just beneath the great trochanter. The pressure of the body above causes the bone to give way at this point so that the trochanters may be higher than the head. In the pelvis malformations are due to the same influence. Here the pressure is exerted on the pelvis in two directions, both from below through the heads of the femurs, and from above acting through the sacrum. In consequence of this the pelvis is laterally compressed, the pubis is thrown forward, and the angle between the pubic bones is greatly narrowed. In addition to this general deformity there is an inward bend in the linea innominata corresponding to the acetabulum. The pressure from above transmitted through the sacrum produces a further tilting of the pelvis forward and a narrowing of the antero-posterior diameter. The tuberosities of the ischium are pressed upward and toward each other. In some cases in consequence of these deformities the cavity of the pelvis may be so reduced as scarcely to allow the natural evacuations through it. In such advanced cases it is impossible for a child to be delivered.

The spinal column undergoes various alterations. The normal curves may be accentuated or new ones produced. In some cases there is a single curve of the column backward, the condition of kyphosis; or this may be accompanied by a compensating curve inward in the upper part of the column, or the curves may be exclusively lateral. The malformations here may be extreme. The thorax is always more or less altered. The soft bones yield readily to the pressure exerted on them by the respiratory muscles. The thorax is compressed laterally and the antero-posterior diameter is correspondingly lengthened, the sternum being pushed forward and outward; these changes are so marked that the sternum frequently has a horizontal position; the ribs show numerous fractures and curves corresponding to the pressure exerted on the thorax. There is frequently a deep depression on each side in the axillary line produced by the weight of the upper extremities on the ribs; the giving way of the clavicle allowing the weight of the arms to bear more directly on the thorax.

Pathological Anatomy

Microscopic examination of the bone may be made either by breaking off splinters of the rarefied bone or by decalcifying it and making sections through it. In some cases the bone may be so soft that sections can be made with the knife without any previous decalcification. Microscopic examination of the bone shows that there are two processes at work. There is an increased absorption of bone without any previous alteration in its structure, the absorption taking place in the same way as in normal bone formation by means of osteoclasts, and a decalcification of the tissue, the decalcified tissue partly remaining in a more or less altered condition, and partly being absorbed. At various places the osseous tissue may be completely absorbed, leaving large areas of fibrous and gelatinous tissue in its place. Where the absorption is taking place the edge of the bone is exceedingly irregular, and the osteoclasts lie in enlarged Howship's lacunæ.

Pommer, as a result of his investigations on osteomalacia, believes that the process consists essentially in bone absorption and that there is little decalcification. Other observers, as Ribbert and Rindfleisch, believe that the decalcification is the main element, the decalcified bone being partly absorbed. Evidences of these processes is given by staining fresh spicules of bone with carmine. Around the edge of these bone spicules there is a reddish zone sharply separated from the rest where the carmine has stained the decalcified portion of the bone. In some cases the bone is very similar in appearance to bone which has been decalcified by mineral acids. Ziegler describes the process as consisting essentially in softening of the bone. Not only is the bone softened on all sides but canals are formed in it running through it. The softening of the bone always takes place from the outside of the osseous beams, and there may be no alterations in the bone structure which still remains, branched bone corpuscles being found in this just as in the normal bone. There is probably little doubt but that this softening of bone does take place in the manner described, and there is also no doubt that along with this there is a process of absorption, without preceding decalcification, by means of the osteoclasts. In all other processes of bone atrophy absorption seems to take place just as in the normal bone by means of the osteoclasts. The bone marrow which remains in the large areas left by the softened bone may be extremely hyperæmic and similar to the fœtal marrow, or it may have the ordinary appearance of yellow marrow. It is probable that alterations in the character of the marrow depend upon differences in the intensity and the rapid advance of the disease.

In all cases of osteomalacia along with the process of absorption and softening of the bones there is in some places regeneration and new formation of bone. The two processes may often be seen in the same splinter of bone and alongside of one another. The growing bone is covered with osteoblasts, and in stained specimens a line may be made out showing the new bone which has been deposited on the old. Where the absorption is taking place the osteoblasts cease and osteoclasts lying in deep pits take their place. That there is a capacity for new formation of bone in osteomalacia is shown by the formation of callus after fractures. The callus which is produced is softer and more porous than ordinary callus, and it is not replaced by true bone as in ordinary fractures. It is probable that the thickening and the deformities of the bones are due in great part to the imperfect callus formation.

A *senile* form of bone change occurring in both men and women has been described by various authors. In this, as in ordinary osteomalacia, there is destruction of the bone with a resulting increased fragility. This condition, although in some respects similar to osteomalacia, should be distinguished from it. There is not the same degree of formation of fibrous tissue here as in osteomalacia, nor is there the same degree of thickening and deformity of the bone. The bones become thin and fragile by absorption and the space is taken up by fatty marrow. Fractures of the bone easily take place, but the soft yielding character of the bone in osteomalacia is not present. True osteomalacia is a disease almost confined to the female and has a close relation with sexual life. Cases have been described in children, but it is probable that there the disease has been confounded with rickets, which it so much resembles.

Roloff has called attention to a *disease in animals* which is in some respects analogous to osteomalacia. The disease is frequently found in beasts which are confined in menageries. It is also found in cattle grazing on marshy lands where the grass is poor in lime salts. Frequently almost the first symptom of the disease is shown by a tendency of the animals constantly to lick one another. They show a great desire for alkaline and earthy substances, and continually gnaw such articles when they are within their reach. According to the rapidity of the disease a variable period passes before other symptoms appear. When the disease has fully developed the animals have some difficulty in standing and their gait is extremely stiff. The bones become so soft that fracture may result from the efforts to arise. Colts appear to be very subject to the disease. They become lame, their bones are painful on pressure, and they show little inclination to move around. This stage of the disease may last from four to eight months. Milking the affected animals causes the disease to progress more rapidly. Oxen do not appear to be affected at all. Here also the bones of the pelvis and ribs are first affected, and the changes are always more pronounced in these than in the other bones. The osseous tissue is reduced in amount, the marrow is mucoid and gelatinous, or it may be hyperæmic and similar to granulation tissue. The Haversian canals are filled up with granulation tissue. In places areas of almost completely decalcified bone, showing little change beyond this, may be found enclosed in the altered marrow. When horses are affected in a minor degree the sway-back or deep saddle results.

Etiology.

Osteomalacia appears to have a more or less well-marked geographical distribution. The disease is most common in Europe, on the banks of the Rhine, and along the rivers leading into the Rhine. In Italy it is most common in the northern part. This has led to the idea that the character of the soil or the climate may be of importance in determining the disease. Kehrer is the chief defender of this theory, and is inclined to group osteomalacia with the chronic endemic diseases.

Winckel has carefully gone over all of the literature on the disease, and concludes that it may appear in any of the countries of Europe and is by no means definitely connected with certain localities. He thinks that the disease is much more common than is usually assumed, and is not always diagnosed.

Sternberg reported two cases of the disease in Norway and says that the idea that osteomalacia is rare in Norway is more a general impression than a fact founded on positive observation.

Kehrer thinks that there may be a certain disposition to the disease which may be inherited, and cites a case in which two sons of a parent suffering from osteomalacia became affected with the same symptoms as the mother.

Kleinwächter thinks that certain races are more disposed to it than others, and finds it exceedingly common among the Jews. He thinks that this is due to the fact that these nurse their children longer than the other races. That this alone is not a sufficient cause for osteomalacia is shown from the fact that in Japan, where the children are nursed by the mother for two or three years, the disease is unknown. Winckel believes that prolonged nursing may have something to do with the disease, but that it acts only as one of many conditions, along with insufficient and improper food, damp dwellings, imperfect ventilation, etc.

The belief has long been held that osteomalacia was essentially a disease of the poorer classes of the community, and was especially associated with bad hygienic conditions, particularly living in damp, ill-ventilated houses. Although there appears to be some relation between poverty and bad hygienic conditions and the disease, it may also appear among the better classes of the community. The greater apparent frequency of the disease among the poor may partly be explained by the fact that poor women often go to hospitals for delivery, and the statistics of the disease largely come from observations in hospitals. Cases occurring in private practice among the better classes are not so likely to be reported. Deficiency of animal food has also been considered as a cause. The disease, however, does not seem to be especially prevalent in those countries where the food of the inhabitants is almost exclusively vegetable. In some countries it is very rare. It is not common in England, although rickets, which is in many respects closely allied to it, is common there. In the United States this disease is extremely rare. Dock, who went over the entire literature with regard to its occurrence in the United States, was able to find only ten cases reported, and in but four of these was the diagnosis confirmed by autopsy.

There have been various theories with regard to the disease, no one of which is sufficient to explain all the phenomena.

Pathology.

In some of the early investigations on osteomalacia lactic acid was found in the cysts of the bone, and the idea has arisen that the disease is due to the destruction of lime salts by means of lactic acid. Lactic acid was found in a few cases in the blood and in the urine. The source of the lactic acid was supposed to be some abnormal processes of fermentation in the alimentary canal accompanied by intestinal catarrh. All of the more recent investigations made on the disease have been against this lactic-acid theory. It has also been supposed that the lactic acid might be due to abnormal processes of nutrition in the bones themselves, the lactic acid being formed in the bones. It has been shown, however, that the lactic acid which is found in the bones at autopsies may be the result of post-mortem changes. On the other hand lactic acid may be present in the blood during life without osteomalacia. There is no experimental basis given for this theory. Dogs have been fed on lactic acid, taking it in extraordinary amounts, and no osteomalacia has been produced.

Recently the acid theory has appeared again in a somewhat altered form. Petrona believes that osteomalacia is an infectious disease which is produced by nitrifying organisms. He has found these organisms in the blood, and has produced osteomalacia in dogs by the injection of cultures of them into the blood. He always found traces of nitric acid in the urine of patients with osteomalacia. According to this theory the lime salts are destroyed by nitric acid which is produced *in loco* by nitrifying organisms. Attractive as this theory seems to be at first sight, it has lacked confirmation by other authors.

In the first place it has been found that this nitrifying property of the bacteria exists only when organic matter is not present or present in but small amounts. Since Petrona's publication on this matter, the blood and tissues have been carefully examined for the bacteria described by him, and his results have not been confirmed. The examination of the urine in a number of cases has failed to confirm the presence of nitric acid which was described by Petrona.

The theory of bacteria could be used to explain the endemic appearance of osteomalacia. It could be assumed that bacteria are present in certain places, either in the ground or in the water, which might enter into the body at the time of pregnancy.

Another theory which has been prominently advocated is that of Rindfleisch. Rindfleisch believes that the disease is essentially due to chronic passive congestion in the bones, and the solution of the lime salts is due to the presence of carbonic acid. The essential nature of the disease according to Rindfleisch is a disturbance of the circulation in the bones, and to this the chronic passive congestion is due. This theory of Rindfleisch has nothing to support it. We know that in the most extreme cases of chronic passive congestion no lesions whatever are produced in the bone. The hyperæmia which is found in the process at its height has much more the appearance of an active than of a passive hyperæmia.

Recklinghausen believes that the disease essentially consists in inflammation of the bone, and he places osteomalacia in the same category with various forms of osteitis.

Virchow, who takes up the question in his article on parenchymatous inflammation, also believes that the condition is one of inflammation, the changes being chiefly degenerative in character.

It does not seem probable that we can place the disease in the same category with inflammations. It is true that at present it is difficult to give any one criterion which shall be considered pathognomonic of inflammation. If we regard the exudation as the essential condition of inflammation we should exclude osteomalacia, for in this there is no trace of exudation.

Von Jaksch was the first to call attention to a diminution in the alkalinity of the blood in osteomalacia. It was at first supposed that this could have some relation to the softening of the bone. A diminution in the alkalinity of the blood, however, is found in a great number of diseases in which there is no osteomalacia, and it cannot be shown to be of any influence in this. There is some agreement between the severity of the disease and the diminution of the alkalinity of the blood. The alkalinity is less when the disease is actively progressing, and increases when it is at a standstill. It has been shown that after Porro's operation or castration, in the betterment of the condition following this, there is an increase of alkalinity.

Cohnheim believes that the essential condition in osteomalacia is the altered relation between the supply and the destruction of the lime salts. He calls especial attention to the fact that osteomalacia is more common in pregnancy when there is the greatest demand made on the tissues of the mother. He also speaks of the analogous condition which is produced in cattle by the use of food poor in lime salts.

The disease has been also connected with abnormal processes in the central nervous system. There is some ground for believing that it is more common in insane asylums than in other places. Pommer and Litzmann have both called attention to this. In some cases the bone disturbances follow shortly after the appearance of various psychoses. Pommer relates a case of combination in a young individual of well-marked osteomalacia with Basedow's disease. He thinks that the congestion in the vessels of the bone is analogous to the retrobulbar congestion producing the exophthalmus, and both are the consequence of arterial congestion.

Neusser thinks that this would seem to show that the disease is related to lesions of the sympathetic. He has investigated the blood in osteomalacia, and finds that there is almost constantly present an increase in the eosinophile cells. The only variation from this was shown in a case of severe osteomalacia in a woman seventy-two years old, in which there was no increase in the eosinophile cells, but there were numerous myelocytes and elements of the bone marrow which do not appear in normal blood. At the autopsy the bone marrow was found red and soft and contained eosinophile cells in small number, but the myelocytes were abundant.

Neusser proposes from this to divide osteomalacia into two forms, one of which is characterized by an increase in the eosinophile cells, and the other by the appearance of myelocytes. The appearance of myelocytes in the blood is indicative of the severest changes in the bone marrow.

Neusser thinks that as osteomalacia presents different phases according to the length of time the process has lasted, and according to the situation and intensity of the process, this may give an explanation of the different conditions found in the blood, and the relation of the blood elements varies accordingly. He has observed one case in which, after successful castration and recovery, there was marked diminution in the eosinophile cells in the blood. He thinks that there is not a relation between the condition of the bone and the ovaries, but a relation between the ovaries and the bone marrow.

Sternberg made a careful examination of the blood in four cases of osteomalacia. He found that there was no increase in the eosinophile cells and did not find myelocytes; making due allowance for the changes in the blood in pregnancy he found there was no change in the hæmoglobinuria nor in the red corpuscles. There appears to be a slight increase in the mononuclear leucocytes.

Relation of Osteomalacia to Diseases of the Ovaries.—It has long been known that there was some connection between osteomalacia and the sexual organs. The disease is one almost confined to women. In most cases it appears coincidently with pregnancy, and there is always a marked increase of the symptoms during both pregnancy and menstruation.

Fehling was led to believe that there was a close relation between the disease and pathological conditions in the ovaries. He was led to this idea by the result of Porro's operation.

Porro in 1878 published an account of a modified Cæsarean section, in which after the extraction of the child the uterus and ovaries were extirpated, and in 1879 Forchier in Lyons expressed the opinion that in puerperal osteomalacia Porro's operation produced recovery by bringing about sterility.

Levy in 1880 expressed himself more definitely in this direction, and Kleinwächter and Sänger, in their successful cases of Porro operation in osteomalacia, ascribe the favorable results to the removal of the ovaries.

Fehling in 1880 published seven cases of osteomalacia with recovery after Porro's operation. Up to that time there had been 44 cases of Porro's operation in osteomalacia. Eighteen of these were fatal and 26 recovered. Of the 26 cases, 3 died later of various diseases, and the results could not be ascertained in 2. In the other 21 cases, that is, all which were investigated, there was recovery or at least a greatly ameliorated condition. Not only were the persons able to walk and capable of work, but the pain diminished, and then disappeared after a few months. There was a marked difference between the result of Porro's operation and that of Cæsarean section. Twelve cases of recovery after Cæsarean section were cited, and in only two of these was there recovery from osteomalacia. Fehling concludes from this that the good effects of the operation are due to the removal of the ovaries. The results of the operation are certainly conclusive. Not only are these results found in cases of osteomalacia occurring in pregnancy, but they are also equally true in the rarer cases of osteomalacia in virgins. Removal of the ovaries here, just as in pregnancy, relieves the condition. The rapid diminution of the pain a short while after the operation is remarkable. Even in two or three days the sensitiveness of the sternum and ribs diminishes, then that of the bones of the lower extremities and pelvis. The process in the larger bones lasts longer than in the thin ones. In one of Fehling's cases the disease was so far advanced that the patient experienced pain even when lying on the back. Two days after the operation the pain was very much better. Usually from two to four weeks elapse before the sensitiveness of the thick bones of the pelvis has disappeared. All accounts agree in the favorable results of oöphorectomy. Fehling found in all of his cases a marked hyperæmia of the ovaries and adnexæ. The hyperæmia may reach the same degree as in the first weeks of pregnancy. Not only are the veins of the pampiniform plexus dilated and more abundant, but the arterial vessels, both of the uterus and of the ovaries, are enlarged. Microscopically he found no marked pathological condition in the ovaries. In some cases they were small and atrophic. In others the tissue appeared to be more cellular than normal, and in some cases there was marked capillary dilatation. He is inclined to believe that in osteomalacia there is a pathological irritation of the vaso-dilators, or a paralysis of the vaso-constrictors of the bones brought about by a pathological activity of the ovaries reflected to the sympathetic. Under the influence of this there is a congestive hyperæmia of the bone in which there is first a solution of the lime salts, then an absorption of the bone tissue. If the constant irritation of the vaso-dilators is removed by the removal of the ovaries, the hyperæmia is diminished and there is a new formation of bone tissue and a diminution of the atrophy. He considers the disease as a trophoneurosis of the bone, first affecting the bones of the pelvis, then those of the thorax and extremities. This hyperæmia of the ovaries and adnexa which Fehling describes has not been found in other cases. Fehling shows that in osteomalacia the fertility of the women is abnormally large. He does not think that the disease is due to the frequent pregnancies, but that the frequent pregnancies arise in consequence of the disease. There appears to be a certain relation between osteomalacia and marked functional activity of the ovaries. Even making every allowance for the good effects which are seen in carrying out most new operations, the results in osteomalacia of oöphorectomy are most remarkable, although it cannot be seen just what the connection between the ovaries and the disease can be.

Latzko thinks that the disease is benefited to a marked degree by chloroform narcosis. He thinks that the good results of the Porro operation and the castration may be due in part at least to the narcosis. He has used prolonged narcosis in cases of osteomalacia with good effects.

Diagnosis.

Sternberg has carefully considered the question of diagnosis in osteomalacia. The history of the patient is usually of importance. In puerperal osteomalacia it is usually typical. There is pain in the back and thighs with difficulty in walking, which appears generally toward the end of pregnancy, ceases after delivery, and in the next pregnancy is increased.

Strümpell thinks that one of the most important things in the diagnosis is the statement which will almost always be made, that the individual has become shorter. When the disease has advanced, and when there have been produced well-marked deformities of bone, the diagnosis is easy. In such cases the alterations of the skeleton are permanent. They are so characteristic that the disease can be mistaken for but few other pathological conditions of the bones. The deformities and the multiple fractures of the bones of the extremities, the extraordinarily rare involvement of the bones of the face and skull, and the marked deformity of the bones of the trunk all call attention to the disease. The body is short in comparison to the extremities. The spinal column in its upper part is generally kyphotic, the thorax barrel-shaped and pressed together laterally so that the sternum has an almost horizontal position. The lumbar vertebræ are shortened, the ribs approach the pelvis, and the abdomen is prominent. Palpation shows many of the bones of the body painful on pressure; some of them, especially the sternum and ribs, bend easily under pressure. In senile osteomalacia the pelvis is free from the disease. In the patients who can still walk the gait is peculiar. The steps are short, uncertain, and laborious. This gait is described as waddling, but it is not absolutely typical of the disease. It is produced by an approach together of the hip-joints, and the slight elevation of the leg in consequence of pain and the weakness of the muscles.

In less advanced osteomalacia with slight deformities of the bone or without any, the diagnosis in some cases is difficult. It can be confounded with diseases of the nervous system, with disease of the joints, and with other forms of disease of the bones. There are some points in common with affections of the spinal column. There is always a constant increase in the tendon reflexes in osteomalacia. The patellar reflex is increased, and there is frequently ankle clonus. There may be more or less paresis, especially of the ilio-psoas muscle. This appears early. Sternberg thinks it is not in consequence of the pain, of the softness of the bone, or of an atrophy from non-use, but that it depends upon a participation of the muscles in the pathological process. A careful investigation of the painful bones is of importance in distinguishing the paresis in osteomalacia from neuropathic paresis. If the spinal column is painful below the end of the spinal cord at the level of the first or second lumbar vertebra, if the pain on pressure extends to the sacrum or the bones of the pelvis, it is much more probable that it is due to a disease of the bones of the spinal column than to a disease of the spinal cord.

In puerperal osteomalacia the symphysis and all of the bones of the pelvis are extremely painful on pressure. Cases of hysteria also may sometimes very closely simulate osteomalacia. The examination of the urine is of little importance for diagnostic purposes. Nencki and Sieber have investigated closely to determine the presence of lactic acid in the urine, and they show that the older reports as to the presence of lactic acid depend upon imperfect methods of observation. The presence of lactic acid in the urine in osteomalacia cannot be regarded as proven. The same holds for albumose. Albumose was found by Virchow in the contents of a bone cyst in osteomalacia. Jones found it in the urine in a case which was regarded as osteomalacia. He was not able to find it in the urine in other cases. Kuhne once found albumose, but did not find it in any other case. Other observers have never found it. Köhler thinks that the albumose is not found in osteomalacia but is found frequently in some of the tumors of bone, as the multiple myelomas which have been mistaken for this.

The microscopic examination of the blood is of no importance in the matter of diagnosis. The results of Neusser's blood examinations have not been confirmed. There may be under various conditions a slight increase in the eosinophile cells in the blood. There may even be some variations in the number of these in normal individuals.

Slight forms of the disease may also have some similarity to peripheral nervous diseases. They may be distinguished from these by a careful examination of the bones. In almost all thediseases of the peripheral nerves the tendon reflex is diminished and in some cases totally disappears, while in osteomalacia it is increased.

The disease may simulate arthritis deformans. This condition may accompany osteomalacia. Affections of the joints may be due to an extension of the bone disease to the joints. Weber has described a severe case of senile softening of the bone in which there was a peculiar hemorrhagic affection of the joints, which he did not regard as a severe form of senile arthritis but as a peculiar synovitis depending on osteomalacia. There is a rather rare affection of the joints which may somewhat simulate osteomalacia. This is a luxation or a separation of the joints of the pelvis. An individual who has undergone this in delivery can have a very similar condition to osteomalacia. It is easy to distinguish the two conditions. In osteomalacia pressure on the pelvis gives rise to pain, while in the other condition it relieves it. In partial dislocation of the bones of the pelvis a tight girdle about the pelvis will relieve the symptoms, while in osteomalacia it produces severe pain. There should be no difficulty in distinguishing the disease from caries.

There may be difficulty in the diagnosis of the condition from tumors in the bone. Primary sarcoma of the bones of the pelvis may simulate the disease, and it is especially difficult to distinguish it from the cases of diffuse carcinomatous infiltration of the bones. This peculiar affection appears to be more common in cases of carcinoma of the prostate than in carcinoma elsewhere. There may also be some similarity to osteomalacia in the cases of malignant lymphoma of the bone. In this there is a multiple formation of tumors of lymphatic structure in the medullary canals of the bones. There is little doubt that this condition has been confounded by some of the older writers with osteomalacia. Köhler carefully investigated the condition and described the clinical differences. The differences distinguishing the two processes, according to him, are, first, that in the multiple myelomas the deformities of the bone are limited to the bones of the trunk. The increased elasticity of the bones is absent and the muscular strength is not diminished. The pains are of a different character from those in osteomalacia. The pain depends upon the growth of the tumors in the marrow spaces of the spongy bone. In certain places it is especially severe. After some days or weeks the pains at these special points will disappear. There is always a great deal of albumose in the urine. In many cases there is a swelling of the lymph glands and spleen.

Treatment.

With regard to the therapy of the disease there is not much to be said. There certainly appears to be some relation, though a slight one, between the disease and unfavorable hygienic conditions. The good effect observed after Porro's operation has been ascribed to the better hygienic relations to which the patients are subjected while in the hospital. This of itself, however, can have but little effect. Not only does the disease appear in the better class of the population, but the better hygienic relations in themselves cannot be shown to be effective. Medication seems to give no results. Various substances have been recommended, cod-liver oil, iodine, etc., but although some favorable influence may be exerted on the general condition of the patient by this medication it does not seem to have any effect on the disease in itself.

Sternberg has administered phosphorus in large doses in four cases with apparently good effects. But in the hands of other observers similar results have not been obtained from this treatment. Castration would seem to be the only thing indicated. What the relation between the ovaries and the disease may be is at present inexplicable. We know that there is a close relation between the various processes of nutrition and certain organs of the body. This has been shown in the case of the pancreas, in the thyroid, and other glands. It is not very probable that Fehling's explanation of the disease as a trophoneurosis due to abnormal nervous influence proceeding from the ovaries is correct. If there is any connection between the disease and the ovaries it is more probable that it is of a chemical than of a neuropathic character. It is possible that other conditions producing abnormal nutrition may be of consequence in osteomalacia. The softening of the bone which takes place in old people, and which has been designated as senile osteomalacia, should be distinguished from the puerperal, as should all forms of atrophy of bone from whatever causes.

HEAT-STROKE.

BY

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HEAT-STROKE:

PROSTRATIO THERMICA—FEBRIS THERMICA.

It is hardly possible to find an entirely unobjectionable generic designation for the affection of which this article treats. So long as it was regarded as the effect of exposure to the direct action of the sun, *Insolatio* was accepted as a satisfactory nosological title, and it was described under its numerous synonyms—*sun-stroke*, *ictus solis*, *ictus solaris*, *apricatio*, *coup de soleil*, *colpo di sole*, *Sonnenschlag*, *Sonnenstich*, *solar apoplexy*, *solar asphyxia*, *morbus solstitialis*, *heliosis*, *febris solaris* or *sun fever*. By others, who regarded the solar influence as causative but desired to indicate the limitation of that influence to the season of the dog-days, when Sirius, the dog-star, rises and sets with the sun, it was called *siriasis* or *siriasis ægyptiaca*. In recent years, when the overheated atmosphere and artificial heat came to be recognized as morbific agents as potent as the blazing sun, it was termed *heat-stroke*, *ictus caloris*, *prostratio thermica*, *coup de chaleur*, *colpo di calore*, *Hitzschlag*, *Wärmeschlag*, *heat-apoplexy*, *heat-asphyxia*, *heat-dyspnœa*, *heat-exhaustion*, *thermal*, *thermic*, or *ardent fever*, according as asphyxia, syncope, or fever was the prominent ante-mortem symptom. Other names of less frequent use have been applied to it, as *phrenitis æstiva*, *phrenitis calentura*, and *erethismus tropicus*. While the one constant pathogenic cause is heat, the phases of the morbid condition induced depend so much on latitude, season, occupation, and idiosyncrasy that the expression *effectus caloris* is perhaps the only one absolutely inclusive of the several groups of dissimilar symptoms, but in view of the suddenness of the invasion in the large majority of cases *heat-stroke* may be accepted as sufficiently generic.

The minor local effects of intense heat, especially that of the sun, upon the cutaneous surface, described as *sunburn*, *ambustio ex calore*, *sole excoctus*, *le hâle*, *caldura*, *erythema solare*, *érythème solaire*, or *Sonnenbrand*: *summer-rash*, *prickly heat*, *lichen tropicus* or *miliaria*: *heat-spots*, *freckles*, *æstates*, *ephelis a sole*, *ephelides*, *taches de rousseurs*, *Sonnenfleck* or *Sommersprosse*, while occasionally severe enough to excite constitutional reaction, do not come within the scope of this paper.

Definition.

By heat-stroke is understood the effect produced upon the body by exposure to intense heat, whether to the direct rays of the sun, to radiation from blasts or furnaces used in industrial operations, or to the overheated atmosphere. The attack or "stroke" is usually sudden, with a very brief, if any, precursory warning, and commonly fatal, unless there be prompt remedial interference. An every-day incident in tropical climates, it also occurs in temperate latitudes, during the prevalence of very hot weather, especially when the hygrometric state of the atmosphere interferes with the normal refrigeration of the body by evaporation from its surface. In the United States its victims are principally out-door laborers, hod-carriers, masons, bricklayers, thatchers, roofers, lightermen, drivers, hay-makers, and others who are overcome while working in the sun, or workmen in sugar refineries, steam laundries, bleacheries, glass-blowers, lime burners, cooks, and firemen in charge of furnaces, or soldiers subjected to fatiguing marches during the hottest time of the day. While the natives of tropical countries are not altogether exempt, unacclimated residents or alien soldiers are there the most frequent sufferers.

Sun-stroke is characteristically a land affection, only rare instances occurring among sailors and those under extraordinary conditions of exposure, overwork, and privation; but in late years, with the enormous growth of steam-navigation, heat-stroke of the most fatal character has become common among firemen, stokers, and coal-heavers, confined in overheated fire-rooms with insufficient hours of relief; and I have very good reason to know that suicide by jumping overboard during the stage of frenzy is frequent among the inadequate engineers' force of passenger steamships.

The statistics of the Medical Department of the Navy of the United States furnish little information on this matter. Prior to the year 1887, there was no distinction made between the effects of solar and those of artificial heat. An average of 14 cases annually was reported under the caption "Insolatio," and of a total of 128 cases in nine years, only 12 were sent to hospital for treatment and 1 died, but it is probable many cases were included under other headings, since in 1886 only 5 were reported, against totals of 23 in the preceding and 42 in the following year. In 1887 cases of sun-stroke and heat-stroke began to be recorded separately, and have amounted as shown in the following table:

YEAR.	INSOLATIO.				CALORIS EFFECTUS.				AGGREGATE.			
	Cases.	Sent to hospital.	Deaths.	Days sick.	Cases.	Sent to hospital.	Deaths.	Days sick.	Cases.	Sent to hospital.	Deaths.	Days sick.
1878	10	..	..	..	..	..	..	..	..	..	..	..
1879	9	..	..	88	..	..	..	..	..	..	..	..
1880	23	6	..	211	..	..	..	..	..	..	..	..
1881	18	3	..	53	..	..	..	..	..	..	..	..
1882	12	1	..	61	..	..	..	..	..	..	..	..
1883	10	1	..	166	..	..	..	..	..	..	..	..
1884	18	..	..	72	..	..	..	..	..	..	..	..
1885	23	1	..	103	..	..	..	..	..	..	..	..
1886	5	..	1	20	..	..	..	..	..	..	..	..
Total, 9 years	128	12	1	774	..	..	..	..	..	..	..	..
1887	12	..	..	71	30	1	..	155	42	1	..	226
1888	3	..	1	3	23	1	..	96	26	1	..	99
1889	1	..	..	1	32	3	..	215	33	3	..	216
1890	5	..	..	44	32	..	..	213	37	..	..	257
1891	5	2	..	29	36	1	..	166	41	3	..	195
1892	6	..	..	12	23	2	1	123	29	2	1	135
1893	3	.	..	55	48	1	..	238	51	1	..	293
Total, 16 years	163	14	2	989	224	9	1	1,206	387	23	3	2,195
Annual average, 1887–93	5	..	..	30	32	..	..	172	37	..	..	203
Average duration of disability	..	..	..	6	..	..	..	5.3	..	..	..	5.5

An aggregate of 387 cases in sixteen years, an annual average during that period of about 22 cases among from ten to eleven thousand officers and enlisted men, serving in every variety of climates, of whom only 23 were ill enough to be sent to a hospital and of whom but 3 died, with a mean period of disability for duty of less than six days, is evidence of the natural immunity of seafarers; but the insufficiency of preventive measures exercised in behalf of the men of the engineers' force is shown by the 32 cases a year of heat-stroke alone occurring since 1887. It is not at all likely that there were no cases of heat-stroke in the navy prior to that year, but its markedly increasing prevalence is a matter of concern. The new nosological table for the Medical Department of the Navy which went into effect on January 1st, 1895, provides for the return among diseases of the nervous system of cases of *prostratio thermica* and *febris thermica*, but it is among the diseases of the circulatory system, the aneurysmata, angina pectoris, cordis dilatatio, hypertrophia, palpitatio, and valvularum morbi amounting to from 27 to 50 cases a year and occurring among men enlisted without physical defect as firemen and coal-heavers, that the more insidious effects of excessive heat upon the organism are to be found. The "irritable heart" of the worker in the fire-room is easily recognized by the medical examiner, and distinguishes the old fireman from the ruddy rheumatic "shellback."

Major Charles Smart, surgeon United States army, the statistician of the office of the surgeon-general of the army, has kindly compiled the following table for me, from which it appears that the mortality from sun-stroke in the army during the past twelve years (5 deaths) has been less than one-eighth that of the preceding fourteen (42 deaths).

HEAT-STROKE OR SUN-STROKE IN THE U. S. ARMY, 1868 TO 1893 (BEING ALL CASES AND DEATHS RESULTING FROM THE EFFECTS OF HEAT FOR THE PERIOD).

Year ending December 31.	Mean Strength.	Cases.	Deaths.	Days sick.
1868	47,472	107	9	Prior to 1887 no computation of time lost was made for separate diseases and injuries.
1869	35,221	44	3	
1870	31,831	41	..	
1871	29,430	55	3	
1872	26,844	44	3	
1873	27,909	36	2	
1874	27,021	17	6	
1875	23,525	30	5	
1876	24,866	42	4	
1877	23,707	43	..	
1878	23,381	113	3	
1879	23,964	31	2	
1880	24,002	21	..	
1881	23,222	54	2	
1882	23,239	31	.	
1883	23,439	22	..	
1884	24,034	30	1	
1885	24,138	49	..	
1886	23,572	29	1	
1887	23,841	45	..	243
1888	24,726	66	1	581
1889	25,008	46	1	611
1890	24,234	73	..	743
1891	23,269	71	..	546
1892	24,203	67	..	314
1893	25,287	43	1	245
Aggregate for 26 years	......	1,250	47	....
Annual average	26,207	48	1.9	....

Average duration of disability, 8 days.

All are not equally prone to succumb to excessive heat. Those enfeebled by previous disease, overwork, or insufficient rest; they who have been subjected to intense mental anxiety or to long periods of privation; the ill-fed, and particularly those scantily supplied with water, the intemperate, unclean, and improperly clad; the occupants of overcrowded insanitary apartments; and generally such as are physically below par are the earliest victims, especially those who carry their disabilities, personal habits, diet, and dress into tropical countries. The tolerance of the acclimatized is largely a matter of education in hygienic common sense. The beer-drinking British soldiers, clad in tight-fitting heavy uniforms, loaded with weighty arms and accoutrements, who were housed, fed, drilled, and marched as in their own little island home, fell by hundreds in India before the warning of the medical officer was heeded, and loose white garments, spacious ventilated barracks, avoidance of exercise during the day, shorter hours of duty, and immediate relief at the first evidence of fatigue, with a carefully regulated dietary and the interdiction of alcoholic drinks, were made matters of sanitary regulation. The history of the old Navy of the United States, when the significantly-termed "sweat-box" was a favorite means of punishment, furnishes similar instances of criminal disregard of the protests of medical officers. Men fell forward unconscious, with burning skins and bounding pulse, when the doors were opened after brief periods of confinement in the coffin-like boxes in which they were compelled to stand upright, and were placed upon the sick-list as suffering from that indefinite affection "adynamia," who would now be registered as cases of heat-stroke, were such now possible under the humane administration which naval hygiene has brought about. Becker has shown that a rise of 1° F. in the surrounding temperature under ordinary circumstances is attended with a rise of one-twentieth of a degree of the body temperature, and Turner relates the result of an experiment on board one of our men-of-war in the China Sea when the temperature of the fire-room at midnight was 152° F. (often exceeded in naval and passenger steamships of to-day) and the body temperature was 98.4° F., pulse 69, respirations 18. After remaining perfectly quiet in the centre of the fire-room for forty-five minutes, the body temperature had risen to 103.6° F., the pulsations were increased to 180, and the respirations to 42, when nausea compelled the medical officer making the experiment to leave the fire-room. At 1:30 A.M., the body temperature was still 100.2° F., the pulse 144, and respirations 30. It is not, therefore, strange that heat-stroke is a common occurrence on board passenger vessels steaming through the Red Sea in the months of August and September, and among both passengers and employés on railroads in Hindoostan. Mr. Planck, sanitary commissioner of Northwestern India, after alluding to a report (June 15th, 1869), that five Europeans had died from heat-apoplexy in one train alone, added: "There is a limit of distress beyond which no living creature can go unhurt, and that limit is reached for a man in a train running against a hot wind or running through a hot land even with the wind."

Symptoms.

The evidences of the ill-effects of heat are not invariable, as the qualifying terms exhaustion, prostration, syncope, asphyxia, apoplexy, and fever, variously applied to them, indicate. They differ in degree and in the organs or functions primarily or prominently affected, these being dependent upon climate, season, occupation, and prior physical condition. The laundress overcome by heat in one of our own cities, the stoker crazed by long hours of labor in the intensely heated fire-room of a passenger steamer, the soldier dropping from the ranks on a march under an Indian sun or seized while asleep in his bunk in a sweltry barrack in the tropics, and the prisoner in a crowded jail, as at Jhang, present very different indications of the disturbed heat mechanism, and heart, lungs, or cerebro-spinal system will each be regarded by different observers as the point of original departure from health.

The characteristic types of the disordered condition due to excessive heat are those exhibiting symptoms of exhaustion, cerebral and pulmonary congestion, and hyperpyrexia, and may be classified for statistical purposes under the heads of (1) *Prostratio thermica*, heat-exhaustion, heat syncope, and (2) *Febris thermica*, heat-apoplexy, heat-asphyxia, and cases which are distinctively febrile.

Heat-exhaustion is a common form in temperate latitudes, and its approach is indicated by gradually developed premonitory symptoms of dizziness, headache, drowsiness, nausea, and faintness, with coldness, clamminess, and pallor of the surface, pupils normal or dilated, frequent, feeble, irregular pulse, sighing respiration, and later complete syncope, terminating fatally unless active medical interference take place, in which case recovery is the rule. The loss of consciousness is never so complete as in heat-stroke proper. In midsummer it is not unusual to see laborers, drivers, and others who have fallen in the streets, and who if judiciously placed in the shade near by and treated on the spot are soon able to return to their homes.

Heat-apoplexy has the characteristics of ordinary apoplectic seizures, for which it may be mistaken: flushed face, injected adnatæ, full, rapid pulse, visibly bounding carotid and temporal arteries, and stertorous respiration. Often there is no premonition, or merely lack of sweating, dizziness, flashes of light or red and yellow or clouds before the eyes. The victim falls and is picked up unconscious, and death, if it be not sudden, occurs after a brief period of convulsions, coma, or asphyxia, unless averted by treatment or should this be ineffectual. Swift found among 60 cases in the New York Hospital that as a rule they had been suddenly seized while at work with headache, sense of fulness and oppression in the epigastrium, occasionally nausea and vomiting, general feeling of weakness, especially in the lower extremities, vertigo, dimness of vision, and insensibility. Surrounding objects appeared of a uniform color, usually blue or purple, in one red, in one green, and in one white. Irritability of the bladder, as Longmore also states, commonly precedes the attack. Wood and others note a peculiar "mousey" odor of the body and discharges. Coplin and Bevan, who had a rare opportunity of studying cases as they were developed in the Franklin Sugar Refinery, found the most constant premonitory symptom and the one of which most complaint was made to be "cramp," usually referred to the region immediately below the ensiform appendix, not infrequently associated with similar pains in the calves of the legs, occasionally in the back, sometimes also in the hypogastrium, less commonly in the thighs and upper extremities, and with it difficulty of respiration as from a weight upon the chest, and sometimes pain in the splenic and hepatic regions.

This is the common form of heat-stroke, and is nosologically the same, the difference being only one of intensity, with thermic fever—the ardent fever of East India writers, which is characterized from the onset by the excessively high temperature (105°–110° F.), hot burning skin, severe headache, pupils contracted to pin-points, thirst, full hard pulse, and dyspnœa. The patient suddenly becomes comatose and dies asphyxiated, unless very active treatment be promptly applied. It is a common occurrence in India that the stertorous breathing or retching of the patient, who is apparently asleep in bed, first attracts the attention of room-mates, who arouse him with difficulty or find him, if conscious, complaining of headache, thirst, and excruciating pains in the body and extremities. In the progress of these cases, the muscular rigidity often amounts to opisthotonos, so great that the body can be lifted by the heels and head. Sometimes it is intermittent with clonic spasms. Usually the sphincters relax, the mere introduction of the thermometer into the rectum provoking watery dejecta.

Surgeon-Major Hume, whose "Diseases of Tropical Climates" is the result of twenty years' continuous intertropical service, makes three varieties of insolatio: 1. *Cerebro-spinal* or *meningeal*, with premonitory symptoms of ardent, stinging heat of skin, great thirst, giddiness, congestion of eyes, nausea, and frequent desire to micturate. "Headache is not very common, but the patient may fall into a state of wild laughter (a very unfavorable symptom); a disposition to commit suicide is manifested in some temperaments; in others he falls insensible; his breathing becomes stertorous and heavy; conjunctiva pinky and congested; the heart tremulous; pulse compressible and feeble; and convulsion and death from asphyxia or apoplexy ensues when coma precedes it" (Maclean). Dr. Morehead considers the following as premonitory symptoms of this form of disease: Headache more or less severe, delirium, tendency to drowsiness, flushing of the face; a hot, dry skin; an accelerated, full, easily compressed and jerking pulse; great thirst; scanty, high-colored urine, and frequent desire to micturate. When the disease has fairly set in, the drowsiness increases, pupils contract, heart's action is tumultuous; pulse very small and easily compressed, skin hot and pungent, muscles undergo convulsive twitchings, respiration hurried and oppressed. 2. *Cardiac;* with no marked premonitory symptoms; the person falls as if by a shock of lightning, becomes extremely weak, and expires from failure of the heart's action, or death by syncope. Sometimes, however, according to Morehead, the effects are not so rapid; severe headache is accompanied with dimness of sight, faintness and great prostration, drowsiness with pupils dilated, a constriction of the chest, sense of epigastric sinking, nausea, sometimes vomiting; skin cold and clammy; lips pale; pulse feeble and slow; the patient gradually recovering or sinking rapidly and dying with convulsions. 3. *Pulmonary Apoplexy;* the patient being picked up apparently drunk, lying in the sun, in a state of coma; pupils dilated and face flushed; death taking place "by coma and by syncope—and causing it to be regarded by some writers as the *mixed variety.*"

Pirrie, a campaigner in Central India in the summer of 1858, also found among the many cases of sun-stroke which came under his observation, three different forms of attack: 1st, The speedily fatal form with only a momentary if any premonitory warning, the victim falling insensible and unconscious of all outward impressions, making a few hurried gasping respirations and instantly expiring. These were cases occurring during direct exposure to the rays of the sun, the redness and heat of the surface of the body, the complete unconsciousness and gasping respiration being the striking features. 2d, The sufferer had an unusual and severe headache, distressing sense of bursting and burning in the eyes, accompanied with giddiness and confusion of vision; a most overpowering sensation of constriction in the chest, with greatly oppressed respiration; great heat of the surface of the body; a dark red, almost livid, color of the skin; and an alarming sense of general oppression and exhaustion. The chief suffering was apparently in the chest, and patients complained most of the symptoms referable to the respiratory apparatus and described them as almost insupportable. If proper means were instantly adopted, consciousness might not be lost and the patient would soon be left to all appearance comparatively well; or the symptoms might increase in severity and merge into the third form in which insensibility supervened upon the dyspnœa and debility. The patient, if seen early, was found in a state of extreme prostration and affected with convulsions, vomiting, burning hot skin, very contracted pupils, excessively suffused conjunctivæ, and rapid feeble pulse. In many cases shortly after seizure, priapism and seminal emission occurred. After a longer or shorter time, the pupils contracted till almost obliterated, the respiration became slower and stertorous, convulsions and vomiting ceased, and the sufferer lay motionless and in a state of low muttering delirium. The skin retained its burning heat but became clammy; the sphincters relaxed, the rapid feeble pulse became more and more weak, and at last the patient died.

Among French authors, Vallin considers that all cases of insolation may be included within two classes, corresponding to what have been described as the sthenic and asthenic forms: the first (asphyxial) in which, after rapid universal heating, the temperature of the blood rises to 45° C. (113° F.) and death occurs, after convulsions, by coagulation of the left ventricle and distention of the venous system; the second (syncopal) where the heating is slower and directed especially upon the nervous centres, the temperature of the blood not being greatly affected and death resulting from disturbance of innervation and consequent arrest of the heart's action as after excitation of the pneumogastric nerve.

Lacassagne, Ball, Dony, and others consider *coup de soleil* (sunstroke) as essentially distinct from *coup de chaleur* (heat-stroke), there being in the latter an enormous and rapid rise of the temperature of the whole body, often brusquely fatal, but generally with prodromes such as nervous erethism, vertigo, photophobia, contraction of the pupils, nausea, vomiting, weight at the pit of the stomach, somnolence, and coma; while in *coup de soleil* the action of the heat is exerted on the point on which the sun's rays impinge—the cranium and brain, the effect being on the nervous centres only, with no considerable elevation of temperature as in *coup de chaleur*. They divide cases into asphyxial, syncopal, and mixed forms. Hestrés says *coup de chaleur* is the result of the pernicious influence of an intense heat, natural or artificial, acting on the whole organism.

Saguet classifies the casualties, which have a thermic origin, as (1) *coup de soleil* or *érythème solaire*, a simple local accident due to the action of the sun's rays on the exposed parts of the body, as the face, neck, chest, back, and arms of lightermen and others, and includes with these cases of what he calls "*coup de soleil électrique*," occurring on board ship as well as on shore among persons exposed to the action of the chemical rays of intense electric light; (2) *insolation*, a grave affection resulting from the action of the sun on the nervous centres, characterized in its mildest form by cerebral congestion, and in the severe form, scarcely seen outside of hot climates, by meningitis and encephalitis; (3) *coup de chaleur* or "*asphyxie foudroyante par la chaleur*," heat-apoplexy, which although observed more frequently in climates of high temperature, is still common enough in our temperate climates when the thermic mean is exceeded; (4) *thermo-héliosie*, a hybrid condition resulting from the association of the two elements of morbid causation, solar radiation and hyperthermia, acting together on the individual, which holds an intermediate place between *insolation* and *coup de chaleur*, its signs partaking at times of one or of the other.

The German writer Jacubasch classes the accidents from heat under three chief heads: 1. *Sonnenstich* (coup de soleil—sun-stroke), the sthenic insolation of Obernier and Vallin, due to the direct action of the sun on the body at rest when the temperature may reach 45° to 46° C. [113°-115° F.]; 2. *Hitzschlag* (coup d'échauffement—heat-stroke), produced by physical activity under temperatures below that of the body, sometimes not greater than 30° C. (86° F.), the form commonly seen in temperate climates among marching troops; 3. *Wärmeschlag* (coup de chaleur—thermic fever), the true heat-stroke of the tropics, resulting from the accumulation of heat in the human economy in consequence of the high atmospheric temperature without direct exposure to the sun or muscular activity.

Strümpell omits any mention of sun-stroke, which, like yellow fever and dengue, is almost unknown in Germany, but Shattuck, in his American edition of the former's "Text-Book of Medicine," recognizes three leading forms of attack, to wit: (1) heat prostration, denoted by faintness, syncope, nausea, vomiting, marked muscular and general weakness; surface of body cool; pulse rapid and feeble; transient insensibility; death from syncope; (2) cases of respiratory as well as circulatory failure due to exhaustion of the nerve centres of respiration and circulation. "Under this head come cases of true sun-stroke, patients who suddenly lose consciousness while exposed to the sun. The skin is cold and the pulse feeble;" (3) cases where the striking characteristic is the great increase in the temperature—thermic fever—with the sequence of symptoms commonly ascribed to this condition—death being due to apnœa and asthenia combined.

Among American authorities, Flint states: "The terms insolation and sun-stroke are applied to an affection, or perhaps more properly, different affections, occasioned, not exclusively by exposure to the sun's rays, as the terms signify, but by the action of great heat combined generally with other causative circumstances. In a certain proportion of these cases, the morbid condition, as judged by the symptoms, is expressed by the term exhaustion; the vital powers are prostrated, the forces carrying on the circulation give way, and death takes place by syncope. In another class of cases, the affection apparently is similar to congestive apoplexy. In still another class of cases the symptoms show a combination of apoplexy and exhaustion—these probably constituting the majority of the cases of so-called insolation or sun-stroke. In other cases the condition is referable to the abnormally high temperature of the blood."

Wood, in his admirable Boylston Prize Essay, says: "There can be no doubt that under the name of sun-stroke or coup de soleil sudden cases of severe illness of very different natures have been described by authors. Such of these cases as have really been dependent upon exposure to excessive heat can be classified under two, or perhaps three, heads, to which the names of *acute meningitis* or *phrenitis*, *heat exhaustion*, and *thermic fever* or *true sun-stroke* may be applied as more or less expressive of the pathological condition existing." The first class, acute meningitis or phrenitis, due to exposure to the sun and the direct action of its rays upon the head, he considers a theoretical class, having never seen or read an unequivocal record of such a case. The second, or simple exhaustion, due to excessive labor in a heated atmosphere, does not differ in its pathology or symptoms from other forms of acute exhaustion, and ought not therefore to be confounded with true sun-stroke.

To be precisely accurate, the expression *effectus caloris* should be used to include all forms of disability from heat, these being classified under the two heads of *prostratio thermica*, heat-exhaustion, and *febris thermica*, heat-apoplexy, heat-asphyxia, the true heat-stroke of Dr. Wood; but the term heat-stroke has come to be in such general use as synonymous with effectus caloris, that it may conveniently, and without very great impropriety, continue to be used as inclusive of cases of exhaustion and syncope as of hyperpyrexia and its results.

I append concise abstracts of typical cases reported by our own hospital physicians and by members of the East India medical service as giving a better understanding of these conditions than the mere collective enumeration of disconnected symptoms.

Exemplifying the class of heat-exhaustion (prostratio thermica) Dr. Wood states that at one o'clock of a hot July afternoon, a man, about fifty years of age, of feeble physique, was brought into his office in a semi-unconscious syncopal condition, with a very rapid fluttering pulse, cool, damp skin and pallid face, who, after rallying, said he had been feeling exceedingly weak and ill for several days. The recumbent position and free administration of brandy caused the pulse to become steadier and slower, and after half an hour he was able to walk with assistance to a carriage.

The senior Professor Pepper in 1851 reported to the College of Physicians of Philadelphia certain cases of sun-stroke, in which the surface during life was cool and in which he found on post-mortem examination the heart very much softened and relaxed, and which Condie, at the time, clearly demonstrated to be simply cases of exhaustion.

Katzenbach reports the case of a merchant, aged thirty-nine, of abstemious habits, who had been closely occupied with business during an unusually hot July, and who, after suffering with pain at the top of the head, went to the seashore, and on August 3d, complaining that his head felt hot, entered the surf, wearing his hat for protection from the sun. After fifteen minutes, he came out without feeling fatigued, and with no other premonition than sharp, darting pains in the head, vomited and fell insensible. When seen his surface was pale and cool, respiration slow and shallow, pupils of moderate size, pulse very feeble and flickering, and axillary temperature 97° F. Consciousness returned after hypodermic administration of brandy and atropine—the temperature ranged from normal to 99.5°, the pulse became fuller, beating from 72 to 88—the headache, intolerance of light, and visual disturbances persisting for some time.

The gradual development of the symptoms, the incomplete loss of consciousness, the cold, clammy skin and subnormal temperature which characterize heat-exhaustion sufficiently distinguish it from true heat-stroke, in which the onset is sudden, the unconsciousness profound, the skin burning hot, and the temperature alarmingly high—the thermometer in the rectum indicating in severe cases 109° to 113° F.

The following series of cases reported by Dr. Townsend, house physician of the New York Hospital, in the *Medical Record* of August 21st, 1880, is typical of the latter condition:

(1) Irish hostler, æt. 45; overheated while working in the sun; 10 A.M., May 27th, drank freely of ice-water; a few minutes later was brought to hospital unconscious; breathing stertorous, with loud mucous rattling in trachea; conjunctivæ injected; pupils finely contracted; skin intensely hot; pulse 158, full and strong; respiration 30; rectal temperature 110° F. Two hours after treatment by ice-cap and cold bath, the temperature fell to 103° F.; patient still unconscious, moaning loudly, grinding teeth, severe clonic spasm of both arms. After alternate rise and fall of temperature, the heart previously strong,

began to fail, œdema of both legs occurred, and at 7 P.M. he died. Temperature 104.7°.

(2) Irish bookbinder, female, æt. 27, poorly nourished, suffered for years with subacute rheumatism, worked in badly ventilated apartment, for two days complained of severe headache, nausea and weakness; May 27th, while at work seized with precordial distress and faintness, obliging her to quit work; half-hour later, vomited freely and fell unconscious; 4:10 P.M. admitted to hospital fully comatose, having automatic movements of left arm, grinding of teeth; conjunctivæ suffused, pupils firmly contracted and insensitive to light; pulse 146, temperature 109° F., respiration 38, labored and noisy; remained unconscious three days, with continued fever; temperature 101°-102° F; great restlessness. On recovery complained of extreme weakness; almost a week before she could sit up; desquamation of entire surface.

(3) Irish laborer, aged 40; June 18th, while exposed to the sun complained of feeling badly and at 4 P.M. fell unconscious; admitted to hospital comatose, convulsive twitchings of left side of face and body; conjunctivæ injected, pupils contracted; pulse 134, temperature 106.8°, respirations 24. After treatment by wet pack, at 7:30 P.M. pulse fell to 99.8°, spasms gradually ceasing; 9 P.M. stupor still continued and attempt to rouse him excited severe tonic spasm, limbs remaining fixed for three minutes, breathing shallow, pulse lost at wrist. 10 P.M. a general tremor lasting ten minutes occurred, followed by return of consciousness. With fall of temperature, improvement progressed, great weakness being the only complaint; discharged after a week's illness.

(4) Irish laborer, aged 56, a strong, well-built man, while exposed to the sun June 25th, at 2:50 P.M., suddenly became weak and fell unconscious; admitted to hospital comatose, breathing rapidly, face and whole body cyanosed, conjunctivæ injected, pupils contracted, pulse 158, temperature 110° F., respiration 40; in fifty minutes after cold bath, temperature fell to 102.4°, pulse ran up to 180, very feeble, extremities cold; and at 4:15 P.M. temperature had fallen to 98.9° and he became very restless; slight twitchings of the facial muscles and grinding of teeth, patient groaned loudly; soon after this a general tonic convulsion followed, head drawn forcibly backward, jaw clenched and extremities rigidly extended, followed by rapid succession of clonic spasms every eight or ten minutes; at 6:30 P.M. pulse 144, temperature 105.8°; respiration 30; wet pack reapplied and at 10 P.M. temperature was 101.8°; convulsions less severe and consciousness returned; at 1 A.M. the following day he was again fully comatose with a temperature of 103° and return of tonico-clonic convulsions; after alternations of rise of temperature to 105° without convulsions, at 4 A.M. of the 27th became conscious and began to recover, but for ten days had to be fed, being unable to use his arms, and did not sit up for three weeks, becoming emaciated, weak, and had severe headache and dizziness for a month.

(5) A German laborer, æt. 42, who had been drinking heavily all day, was found at 5 P.M., June 27th, in his room unconscious; admitted to hospital comatose; pupils contracted; pulse 144; tempera-

ture 108.6° F.; respiration 38, loud, stertorous. Respiration ceased upon immersion in bath of 75° F. but in a few minutes was artificially restored; temperature fell to 105°, but at 6:40 P.M. the man died of failure of heart's action.

(6) An American laborer, æt. 22, admitted 8:05 P.M, June 28th, unconscious, breathing stertorously with loud rattling in trachea, face and body cyanosed; profuse frothy mucus issued from nose and mouth; extremities cold; rectal temperature 108° F.; no radial pulse; intense pulmonary congestion, œdema; the heart did not respond to large cold-water enemata and hypodermic injections of brandy and digitalin, but the temperature fell in forty minutes to 105.8°; ten minutes later after clonic convulsions, marked opisthotonos, and involuntary ejection of contents of bowels and stomach, he died.

(7) Irish waiter, aged 40, who was required to remain nearly all day in a kitchen where the thermometer stood at 100–101°, after having complained for two or three days of headache, dizziness, and prostration, fell unconscious on June 30th at 8:30 P.M.; temperature 107° F.; wrapped in wet sheet, rubbed with ice and sent to hospital; admitted comatose, pupils contracted, breathing stertorous; at 9:30 P.M. pulse 144, temperature 102.4°, respiration 30; placed in bed and immediately began to have convulsive twitchings of the upper extremities, which grew severe and were accompanied by loud moaning and grinding of the teeth; 11 P.M. recovered consciousness, complained of great weakness, but in three days was able to be discharged.

(8) An American porter, æt. 30, after working hard all the morning of July 12th in the sun, walked a short distance, complaining of dizziness, confusion of ideas, and weakness and fell unconscious; admitted 2:30 P.M., comatose, clonic spasms of the whole body; face pale; eyes suffused; pupils contracted; pulse 140, temperature 108.2 F., respiration 38; after two hours in the cold bath, temperature fell to 101°, and half an hour later spasms ceased, and consciousness returned; four days later there was desquamation of cuticle of chest and abdomen; discharged a week after admission.

(9) An American carpenter, aged 45; was seen to fall in the street at 3 P.M. on July 14th; admitted to hospital 5 P.M., comatose, breathing stertorously, skin intensely hot, pulse very rapid, temperature 109° F., respiration 35; signs of œdema of lungs appeared; temperature, after falling under ice cap and cold bath to 108°, rose to 110.2° at 6 P.M.; after reapplication of ice pack, temperature 102.2°; 7 P.M. still comatose, respiration loud; tonic rigidity of both arms and muscles of neck, drawing head far back; 7:30 P.M. temperature fell to normal, pulse became lost, patient sank into a state of collapse and died at 9:15 P.M. with a temperature of 103.4°.

The above series of cases admirably represents the general character of the severer form of heat-stroke as seen in the summer season of our climate, as well as the various differences in degree and sequence of symptoms due to nationality, age, temperament, occupation and exposure, and may be appropriately supplemented by the following from another hospital and in another year, as reported by

Dr. Freeman of the Presbyterian Hospital, in the issue of the *Medical Record* of New York of November 24th, 1883.

(10) An unknown laborer prostrated by heat was brought to the hospital at 11 A.M., June 13th, unconscious, face pallid, eyes turned up, pupils contracted and insensible, imperceptible radial pulse, rectal temperature 113° F., respiration 28, very shallow; after thirty minutes' application of wet sheets and ice to the head, a very thready pulse of 188 could be felt at wrist and in an hour the temperature fell to 106°. At 1 P.M. it had fallen to 103.5°, pulse 155, respiration 28; the man vomited and had a loose fæcal passage; at 1:30 temperature 99.5°, pupils alternately contracted and widely dilated; slight convulsive movements began to appear in both upper and lower extremities and occasional loud inarticulate sounds uttered; pulse grew weaker; pupils dilated widely; convulsions increased in severity; 4 P.M. temperature rose to 104° and, notwithstanding cold applications, to 106° and then fell to 103°—violent tonic and clonic spasms occurred every few seconds; finally pulse disappeared, face became cyanosed, and death occurred at 6 P.M. in a violent convulsion.

(11) Irish laborer, æt. 20; admitted 5:30 P.M., June 7th, unconscious, having general convulsions and frequent vomiting; pupils contracted; pulse 166, feeble; respiration 36; temperature 109° F., rising notwithstanding ice-cap and ice-water to surface to 109.5°—falling in an hour to 106.3°, when convulsions ceased, and forty minutes later to 101°. An hour later it had risen to 106.8° and after renewal of cold-water treatment and hypodermic administration of whiskey fell to 101.2°, when he began to improve with return of consciousness; vertigo persisted for two weeks.

(12) Irish laborer, aged 56, admitted 12:50 P.M. on July 17th, unconscious, pulse 128, full, soft; respiration 28, stertorous; rectal temperature 109° F. In fifty minutes, temperature was reduced by cold applications to 101° F.; soon after slight twitchings were noticed in both upper and lower extremities, and he began to groan loudly with each respiration; vomiting occurred and temperature rose to 102.2°; four hours later the man became conscious; no further rise of temperature occurred, but intellect was considerably impaired; on second day diarrhœa set in and delirium came on at night and the next day was violent, the patient requiring restraint; after this he slowly improved and by August 1st had completely recovered.

Dr. Richards reports 5 cases treated at the Rhode Island Hospital in July, 1887, occurring among laborers (Italians), who were all found unconscious, with contracted pupils, very rapid feeble pulse, breathing stertorously and having rectal temperatures ranging from 108.5° to 110°—of whom one died in fifteen minutes, one in a few hours, and a third not until after eight days, whose temperature at seizure was 108.5° and at death 106.8°. The two who recovered had a temperature of 109°.

Dr. F. A. Packard reports 31 cases of laborers, bakers, street-pavers, sugar-refiners, and 1, a waiter in a comfortable club, who were brought to the Pennsylvania Hospital, in the summer of 1887, prostrated by heat, of whom 12 died, 1 having had a temperature of 108°, 2 of 109°, 1 of 109.2°, 6 of 110°, 1 of 111°, and 1 of 112°. Of the 19 recoveries, 1 had a temperature of 100°, 1 of 101°, 1 of 102°, 2 of 103.4°, 1 of 105°, 4 of 106°, 1 of 106.2°, 1 of 106.4°, 3 of 108°, 1 of 108.2°, 2 of 109° and 1 of 110°—or 13 of and above 106°. The pupils were contracted in 24, normal in 5, sluggish in 1, and dilated in 1. When contracted, there was also unconsciousness, with temperature above 106°. Convulsions occurred in but 6 cases; they were never epileptiform, but usually tonic contraction alternating with clonic spasms. The case of dilated pupils occurred in an Englishman, a clerk, whose intemperate habits had reduced him to the position of a laborer. When seen he had a rectal temperature of 110°, was unconscious, breathing stertorous, with pupils dilated and equal, but which two days later became contracted. The Swiss clerk fell unconscious while waiting at a club-room table at 9:45 P.M. and was picked up pulseless, breathing stertorously, pupils contracted, livid, blotchy facial appearance, and having a typical mousey odor. There was a profuse semi-liquid bowel movement. The temperature was 112°, which was reduced by ice-bath in forty-five minutes to 101°, but rose again to 109° at 4 A.M., when he succumbed.

In a clinical lecture delivered by Dr. Wood at the Hospital of the University of Pennsylvania, he called attention to the peculiar odor of sun-stroke, which he says is "as distinct and as unmistakable as is the odor of typhus fever, small-pox, or any other disease." It is especially marked where there have been involuntary evacuations, the latter losing their true fæcal smell. The case presented in which this odor was marked was that of an intemperate Irish laborer of twenty-two, who while at work in the grounds after a protracted spree began to feel weak and dizzy at 10 A.M. and fell unconscious at 11:30, vomiting incessantly, having frequent involuntary stools, slight twitching of the general muscles increasing to decided but not severe convulsions, with marked blueness of the surface and almost complete arrest of capillary circulation. The rectal temperature of 110° F. was reduced in an hour and a half by application of ice to 102°, with ultimate recovery.

The several cases above described sufficiently illustrate the affection as it is seen during the heated terms of our own latitude. The following, reported by Surgeon-Major William Curran, are typical of the tropical heat-stroke of British India:

(1) A sergeant, æt. 23, muscular, rather bloated-looking, but not of actually intemperate habits, after recent over-indulgence, was apparently in perfect health on the evening of June 1st, at Cawnpore,

and was seen smoking on the veranda of his barrack-room about 3 P.M. He had, however, been heard to make some remarks about not feeling quite well, and toward noon complained of headache, drowsiness, and nausea. After lying down to sleep off his uneasiness, his breathing was so stertorous and his face so livid as to attract the attention of his comrades, by whom he was sent to the hospital, where he was admitted unconscious, dying fifteen minutes later. Ten hours after death the face was suffused and puffy and the axillary temperature still abnormally high.

(2) A private, aged 37, a healthy man of temperate habits, was admitted to the hospital at Futtehgurh on May 29th, with fever, hot dry skin, insatiable thirst, and severe headache. After conversing with his wife on the veranda, he returned to his couch and was soon (8:45 P.M.) seized with a sudden feeling of nausea and faintness; subsequently he had a severe convulsion, during which he became unconscious; conjunctivæ bloodshot; pupils contracted and insensible; respiration labored and sighing; pulse slow, compressible, and disposed to fail. In spite of cold-water treatment, stupor became profound at 2:30 A.M., respiration and circulation progressively feeble, and at 5 A.M. he died, eight hours after seizure.

(3) A private, 31 years old, a small but healthy man, of temperate habits, who had been only seven months in India and had ailed with a slight fever a few days previously, was brought to hospital at 8:30 P.M., July 1st, having been attacked while preparing his bed for the night at the barracks. Skin burning hot; conjunctivæ suffused and muddy; pupils slightly dilated and insensitive; respiration noisy and puffing; moaned continuously; lips covered with white frothy saliva; roused from stupor to utter short monosyllabic grunt. The cold douche was resorted to, the temporal artery on one side and brachiocephalic vein on the other opened without flow of blood from either. After an interval of apparent improvement, the dyspnœa increased, conjunctivæ became more injected, the pupils contracted to pin-holes, and he died thirty-seven hours after seizure. Seven hours after death, the body-heat was found abnormally great and persistent and muscular rigidity was marked.

(4) A private, æt. 26, admitted to hospital at Peshawur, 1:20 A.M. of July 19th; very pale and livid, pupils contracted, surface intensely hot, respiration stertorous; pulse 140, small, very compressible; involuntary fæcal passage. After cold douche, temperature fell at 3 A.M. to 100.1° F. and an hour later to 97°, after vomiting and involuntary stool; at 6 A.M. moaning plaintively, face ghastly pale, lips compressed; at 10 A.M. temperature had fallen to 96°, respiration became calmer; the man started when spoken to. Next day he was conscious and free from giddiness and headache, and apparently improved until night of 23d, when he rambled, pulse ran up to 126, and respiration became rough, noisy, and bronchial. Although pneumonic complication was evident he was reported convalescent on the 28th, but on the 30th the right lung became involved, pulse was small, weak, 112, and he died August 4th of exhaustion.

(5) A gunner, aged 32, who had been in hospital since July 11th with a low form of remittent fever, complained of headache on the

26th, and at 4 A.M. of the 27th, the weather being very close, sultry, and oppressive, cried out for a coolie and immediately became unconscious. When seen face had a collapsed, pale, leaden hue; eyes deeply sunken; conjunctivæ injected, pupils contracted; skin harsh, dry, burning hot; pulse 140, very small, compressible; respiration oppressed. 7 A.M. became conscious, pulse 148, temperature 103° F., soon reduced to 102.4° by cold douche, while the pulse became imperceptible; convulsive twitchings persisted. At 10 A.M. notwithstanding exposure to the cooling blast of a thermantidote and ice to head, temperature was 105.4°, pulse 136, convulsions more frequent and protracted; at 11:30 A.M. no pulse at wrist; lapsing into coma; temperature 107.2°; convulsions recurred with great severity. Died at noon. Six and a half hours after death axillary temperature stood at 107°.

(6) Mrs. ——, aged 35, who had been admitted to hospital August 6th, with harsh, dry skin, thirst, and headache, the result of previous illness, was found at four o'clock on the morning of the 14th unconscious, pupils not contracted, convulsive movements of head, face and extremities, moaning incessantly; respiration stertorous; temperature 108.4°, pulse 160; former reduced by cold douche to 99°, and the pulse to 100–120. At 10 A.M. temperature had risen to 103°, pulse was 133, and a slight return of consciousness; but at 1 A.M. of 15th, the pulse was 150, temperature 104.6°, and patient died from exhaustion at 8:30 A.M.

Dr. MacRoberts of Newcastle, New South Wales, claims the Australian type to be different from the Indian, but the differences are only such as are due to climatic modifications of physical impressibility, permitting a longer preliminary period of resistance and less abruptly fatal result. Severe pain in the head, delirium at night, constipation, vomiting, convulsions, aphasia, and hemiplegia are not characteristic of a distinctive type. The several prodromes, symptoms, and sequelæ of the affection are given prominence according to the stage of the attack in which the patient is seen by the reporter, its severity, and the latter's practical familiarity with its clinical history; he, perhaps, is also influenced by the prevalent tendency to discover new phases of morbid action, which has been the same since man has been subject to the physical laws of his environment. "Little that is absolutely new," says Surgeon-Major Curran, "can be said of a disease that has been known since the days of the Shunammite and that distributes its favors so equally to the poor as well as to the rich—in the mud hovel as in the palatial hospital or barrack-room." Persistent dry heat is the main factor in its production. The effects of heat so applied are cumulative and after a greater or lesser period sometimes inappreciably brief—the "stroke" occurs, the victim falling unconscious while at work, or on the march, or being discovered comatose and stertorous when supposed to be quietly asleep in bed. Other circumstances than mere temperature conduce to this result. Lithgow, the traveller, "saw smiths work horseshoes and nails out of cold iron, which is only mollified by the vigorous heat and rays of the sun and the hard hammering of hands upon the anvil." I have myself walked under a mid-day sun at Manilla with the shade temperature at 120° F., unscathed and without discomfort, and again at Hong Kong and Canton at the hottest period of the hottest days of the tropical year. The ancient Greeks walked bare-headed in mid-summer as do the Russians of to-day, and the so-called human salamanders remain, for exhibition purposes, in heated ovens while they cook eggs. Sun-stroke is more common in New York than in New Orleans, through neglect to adapt habits to the physical conditions that are exceptional in the one and persistent in the other. In the words of Arnold: "Drink has cost England more graves in the East than all our wars since Clive." Three years used to be the average duration of European life in India, but to-day the ordinary European avoids as much as possible exposure to the hot weather, and as to the soldier he is rarely allowed any discretion in the matter. In our own climate, the ignorant laboring class, imprudent in dress and alcoholic indulgence, or whose necessities compel excessive or untimely toil, are the chief sufferers.

There is another class of helpless victims of this affection who must be taken into account. It is interesting to note that one of the earliest recorded cases of insolation was that of the child of the Shunammite woman, who "went out to his father to the reapers. And he said unto his father, My head, my head. And he said to his servant, Carry him to his mother. And when he had taken him, and brought him to his mother, he sat on her knees till noon, and then died" (2 Kings iv. 18–21). The subsequent recovery of the child under Elisha's treatment, which physicians of these days cannot hope to practise so successfully, is told in the succeeding verses (33–35). Illoway has recently shown that heat-stroke (thermic fever) resulting from overheated apartments must be considered in the category of the summer diseases of infants, and suggests the probability that they have hitherto "remained unrecognized, being engulfed in that great vortex that is made to swallow so many things, to wit, cholera infantum." He relates several cases that may be considered typical:

(1) A nursing year-old baby, in good flesh, which had been seized on a hot September afternoon with vomiting and diarrhœa, was seen unconscious with feeble, scarcely perceptible pulse, filmy eyes, shallow gasping respiration; muscles of jaw firmly contracted; the body, especially occiput, hot and dry to the hand; axillary temperature 106° F.; face purplish and having the appearance of asphyxia. The child recovered under hydriatic treatment.

(2) A well-developed nursing infant, aged 10 months, was taken ill on an intensely hot June night with vomiting and diarrhœa and was found with eyes half-closed, moaning occasionally and restless; axillary temperature 104° F., which rose despite frequent spongings to 105.7°, with great heat of head, excessive restlessness, hurried respiration, marked movement of the alæ nasi and rapid pulse, which were relieved on the application of the cold wet-pack.

(3) A bottle-fed baby, a year old, living in a badly ventilated apartment, was seized with vomiting at 10 P.M. of a hot summer night, and became restless. The head was hot to the touch, eyes dull, face pale, axillary temperature 103°; pulse about 130. It was supposed to be a case of incipient brain trouble, but with the idea that the heat of the room might be responsible for the condition of the child, the application of cold cloths was directed with the prompt reduction of temperature and relief of the other symptoms.

Surgeon-Major Hume of the Indian Medical Service reports a case of what he considers *local insolation*, occurring in an officer, thirty-three years of age, of nervous temperament, the left side of whose head and body, especially the neck, was exposed to a hot Indian sun the whole day in a march, and who on arriving at camp, felt unwell and very hot. He took a bath, somewhat relieving his discomfort, but later, on going to mess to dinner, after taking some soup, had an attack of vertigo and faintness and reached his tent with difficulty. At 9 P.M. he was found with very slow (about ten per minute) labored, apparently asthmatic respiration, the dyspnœa especially marked on left side; pulse very slow and at first scarcely perceptible; nausea; excessive headache, particularly on right side, expression anxious, lower extremities in constant motion; face and body pale, frequent yawning. In Dr. Hume's opinion the cause of the symptoms, though unusual, was unquestionable, the direct action of the sun on the exposed left side of the neck having caused heat-stimulation of the vagus, giving rise to inhibition of the respiratory and cardiac movements and consequent dyspnœa.

Causation.

There have been various explanations of the modus operandi of high temperatures. Tyndall says: "The heat of the blood is scarcely affected by an augmentation of the external heat, as this heat instead of being applied to increase the temperature of the body is applied to change the aggregation of it; it prepares the perspiration, forces it through the pores and in part vaporizes it, hence it is that under the varying conditions of climate the temperature of the human blood is practically constant. The blood of the Laplander is sensibly as warm as that of the Hindoo, while an Englishman in sailing from the

North Pole to the South finds his temperature hardly heightened by his approach to the Equator and hardly diminished by his approach to the Antarctic Circle." This is the case under normal conditions, and so long as it is true, heat-stroke will not occur. Why it does occur has been the subject of much speculation.

Professor Stiles, then a medical officer of Hancock's division, observed that on the march of the Union army from the Rappahannock to Gettysburg in June, 1863, when cases of sun-stroke were frequent, the symptoms pointed to the heart rather than the brain as the seat of the disease, and as a result of experiment on the similar pathological condition artificially induced in lower animals, demonstrated that the blood itself is not injuriously affected by a heat of 115° F., that the nervous tissue is left unharmed by a temperature that will paralyze the muscle—but that a mammal cannot long survive a temperature of 115° of the blood in the left side of the heart, and when exposed to a heated medium long enough to produce this result it dies with the phenomena of heat-stroke. He attributes the fatal result to paralysis of the muscular tissue of animal life by a heat of 115° F., for which he quotes the corroborative authority of Edwards and Bernard. Sun-stroke he considers a febrile disease. While the direct effect of a temperature of 115° suddenly applied to non-striated muscular fibre is its rigid contraction, if gradually applied the most complete relaxation precedes the *rigor mortis*. The febrile phenomena, he asserts, are due to the paralysis of the blood-vessels throughout the body; the blood is poured from arteries into veins; the heart is aroused to tumultuous action hastening the period of its own complete exhaustion or paralysis; the brain is overwhelmed by the torrents of blood in its dilated vessels; and the "stroke" marks the period of vascular paralysis and of the release of the muscular tissue of the vessels from the control of the sympathetic nerves.

Vallin, after extensive experimental investigation of the effects of high temperature on animals, concluded, because myosin coagulates at the temperature of 45° C. (113° F.) and because in post-mortem examinations after heat-stroke the left ventricle is found contracted as hard as wood and its cavity almost effaced, and there are no traces of spontaneous pulsation of the heart, which is also completely insensible to the electric current, while after death from other accidents the heart continues to beat an hour or more and under electric stimulus several hours; that, therefore, the principal lesion from which all others result is the arrest of the heart's action from sudden rigidity of the left ventricle and diaphragm, which loses its sensibility next after the heart, due to the coagulation of their myosin. Accompanying this rigidity, Hermann has shown there is a strongly acid reaction of the muscular tissue, denoting the death of the muscular element and the total loss of its physiological properties, and this nowhere more marked than in the left ventricle.

Incidental reference may be made to Professor Huguenin's statement that "recollecting that experiments have shown that moderate heat, directed upon the cranium, causes dilatation of the vessels, we must conclude that the initial hyperæmia of sun-stroke is due to heat, but this, however, is the least of the changes produced by insolation, for though, at the onset of the symptoms, there is great hyperæmia of the pia and brain, after death Arndt shows extreme anæmia of both is observed, with great distention of the whole venous system;" and Professor Nothnagel says: "The assumption that the direct impingement of the sun's rays upon the head may excite an active congestion is unwisely accepted as a fact, but it has been placed beyond doubt that the direct influence of the sun has nothing to do with the production of the group of symptoms characterized as sun-stroke or heat-stroke. The hyperæmia discovered after death is venous in character and a secondary phenomenon immediately dependent upon the diminished power of activity of the heart, and Obernier has shown by clinical and experimental observations that the cause and danger of sun-stroke are to be sought in the abnormal increase in the temperature of the body."

Hutchinson, almost alone, does not regard the theory proven which places sun-stroke in the class of fevers, for while many symptoms and lesions are common to this and to febrile conditions, the same is true of other states which are confessedly not fevers.

Pertinent to the elevation of temperature during fever, Professor Hertz of Amsterdam says: "It is not merely a matter of increased production of heat and diminished loss of heat, but we have to do with a modification or disturbance of the power that *regulates* the heat of the body, by which disturbance, according to Liebermeister, the average grade of temperature is raised to a higher point than normal, and whether the special centre regulating the animal heat is an excito-caloric or a moderating centre or possesses a double character, it is certain that in fevers this nerve-centre is so affected that it is disturbed in its functions. A certain number of the manifestations that accompany the fever, headache, dizziness, malaise, violent trembling, convulsions, etc., must be regarded as results of the increased supply of heat in the body and as the effect of the heated blood on those nerve regions."

Excessive heat is undeniably the morbific agent in the condition under discussion, and whether death occur from syncope, coma, or asphyxia, it is the consensus of opinion that the apparatus of innervation is the channel through which the vital powers are assailed, and that the proper place of heat-stroke (prostratio thermica and febris thermica) is among the neuroses. The rationale of the pathological process cannot be more clearly, tersely, and plausibly stated than in the language of H. C. Wood, as his conclusions from repetitions of the experiments on animals of Stiles, Kühne, Claude Bernard, and Vallin, and from others of his own: "When a man is exposed to heat beyond his powers of resistance, there is a gradual slow rise of temperature, until the stimulus of the heat becomes so intense as to paralyze either the heat-centre or the vaso-motor nerves, as the case may be, and then there is probably a sudden intensifying of the oxidation processes, and a further rise in temperature, preceded, accompanied, or followed by an overwhelming of the cerebrum; in other words, intense fever, with its accompaniments, is developed," whence the name *thermic fever* given by him in 1873—or *heat fever* proposed subsequently by Prof. George B. Wood.

Morbid Anatomy.

The morbid anatomical appearances revealed by necroscopies depend greatly upon the time after death when the examination is made, the temperature of the cadaver often continuing for hours above 100° F., accelerating putrefactive changes in the blood and parenchymata and loss of the muscular rigidity, which had led to the belief that coagulation of myosin was the cause of death. Pirrie states that on account of the extraordinarily short time between death and interment in Central India, autopsies were not common, and that among the suddenly fatal cases scarcely any morbid changes have been observed, and it was a remarkable circumstance that these were far from being in proportion to the urgency and rapidity of the symptoms. Engorgement of the scalp and conjunctivæ and turgescence of the pia mater were noted, and sometimes engorgement of the lungs to the extent of almost blackness in color was a striking appearance. In one of Longmore's comparatively slow tropical cases, there was extensive engorgement of the lungs with obstruction of the pulmonary circulation, some cerebral congestion and engorgement of the vessels of the pia mater and choroid plexus, without serous or sanguineous effusion in the cavities, and in a similar case reported by Murphy the cerebral veins were full with slight congestion of the posterior parts of both lungs.

Wood also states that the post-mortem appearances are mostly negative, there being scarcely any constant lesion. Perhaps the most notable in cases examined *immediately* after death, in our own climate, is rigid contraction of the left ventricle, its cavity being empty and almost occluded, the right side of the heart and pulmonary arteries containing a small amount of dark fluid or partly coagulated blood, which is feebly alkaline or decidedly acid, the corpuscles crenated with diminished tendency to form rouleaux. The blood appears to have left the arterial system and to have collected in the venous trunks. As a rule the large vessels of the pia mater are full of blood, with more or less congestion of the cerebro-spinal meninges generally and of the brain slightly, and both it and the cord are softened; the lungs dark red, almost black, sometimes hemorrhagic, sometimes œdematous; sometimes the spleen is swollen and soft, and sometimes there is parenchymatous degeneration of liver, kidneys, and heart, the latter usually dark in color. Ecchymoses, subserous hemorrhages and extravasations of blood in and around the superior sympathetic ganglia, vagus, and connective tissue are also occasionally seen, and according to Köster, swelling, hemorrhage, separation and destruction of the nerve fibres and extravasations in both vagi and phrenic nerves. Loss of coagulability of the blood is the one great lesion of human *coup de soleil* to the existence of which all observers bear witness, and consequently it is highly improbable that death is ever due to the formation of coagula during life. The cases reported by the senior Professor Pepper in 1851, in which the heart was very much softened and relaxed, were shown by the coldness of the surface to have been simply heat-exhaustion.

Prophylaxis.

Sun-stroke is commonly preventable by proper precaution. While the necessities of laboring men compel them to work under all conditions, they should be admonished against the black felt hats, thick flannel shirts, heavy trousers, and unnecessary waistcoats and jackets with which Irish hod-carriers and other laborers clothe themselves in the most sultry weather. The practice of some city Boards of Health should be general, of issuing warnings to the working classes against close, unventilated sleeping-rooms, the abuse of stimulants, and working without shelter from the sun, and of advising the placing of constantly wetted cloths and green leaves inside the hat, with the drinking of cool water, tea, or coffee to furnish material for evaporation from the surface. Better ventilation of ships' fire-rooms, shorter periods of duty, and medical supervision of firemen, coal-passers, and stokers coming from watch will prevent much of the heat prostration among this class. There is no more important duty of the naval and military medical officer than the prohibition of exposure, injudicious clothing, and improper diet of both officers and enlisted men. Surgeon Smart of the United States Army was able during a service of four years in Arizona, with a climate almost as hot as that of India, most of the time in the field with commands of from fifty to five hundred men, making long marches often on scant allowance of water, to escape with but one case of sun-stroke, by his care that the men should always have some water available; and Major Smart states: "In the one instance where sun-stroke disabled the command this rule was not followed." When the skin becomes hot and dry and sweating ceases, the medical officer should at once order relief from duty, rest, and a cold bath. As attacks of heat-stroke commonly follow heavy meals, the diet should be restricted, especially when there have been premonitory dizziness and malaise, alcoholic stimulants avoided, and the surface sponged with cold water or sprayed with alcohol or diluted vinegar. In the tropics, the clothing of the natives, which is light and loose, except the head-dress, which fits closely, should be imitated as far as possible. The rapidly moving railroad trains in India, even those running at night, create hot blasts, similar in their effects to the simûms and siroccos, dreaded as "poison-winds," and while the exigencies of the public service may not permit the absolute discontinuance of all travel in overheated seasons, the periods of relief of employés from duty should be very much increased and their runs shortened.

Treatment.

There is substantial agreement as to the treatment of heat-stroke. Although there can be no absolutely invariable routine of procedure, it is important there should be no undue haste in the removal of the patient, and remedial measures should be begun upon the spot, whether the case be one of mere thermic prostration or of the sthenic form of the condition.

For the former the treatment for ordinary syncope suffices. The supine position in the shade, removal of all constricting clothing, the inhalation of ammonia vapor or of sprayed alcohol—diffusible stimulants, such as aromatic spirit of ammonia or small doses of whiskey, or if there be nausea and vomiting their hypodermic or rectal exhibition, will induce the heart's reaction and permit the patient's removal to his own home. Cases of more profound prostration may require external stimulation by mustard, flagellation, enemata of turpentine, and hypodermic use of digitalis and strychnine.

Cases of heat-stroke proper are greatly aggravated by neglect of immediate treatment and by being hastily put into an ambulance and jolted off to a hospital, when a few buckets of cold water, ice to the head, or prompt venesection would have contributed to recovery. Excess of heat being the morbific agent, the manifest indication is to reduce that abnormal heat as quickly as possible, and the easiest and most effectual way of accomplishing this is by the application of cold water, which not only directly abstracts heat but by absorption furnishes material for refrigeration through evaporation. In all well-equipped hospitals there are proper facilities for cold affusion, cold baths, Kibbee cots, Stephen's hammocks, Walton's beds and other devices, ice packs, douches, sprays, or cold-air blasts, but Ellis of New York, an experienced ambulance surgeon, has shown that it is as senseless to postpone treating sun-stroke until the hospital is reached as it would be to delay to secure a severed artery, and he insists that every hospital-ambulance should be supplied with ice, a pail, and a water-sprinkler, so that the treatment begun on the spot shall be continued during the transport of the patient to the hospital; and he instances the case of an Italian picked up unconscious, with vomiting and involuntary passages and having a temperature of 108.6° F., who was freely sprinkled with ice-water in the ambulance and reached the hospital in forty-five minutes with his temperature reduced to 101° F., and recovered.

Whether the patient be placed in a cold bath or wrapped in wet sheets, or be packed in ice, or be rubbed with lumps of it, care must be taken not to reduce the temperature too far. When it falls to 102° or 103°, he should be removed to a dry bed, rubbed dry, and wrapped in warm blankets, being disturbed as little as possible in the operation; and as the temperature rises again, cold towels or ice-bags should be applied, cold-water enemata administered, or the bath, pack, or douche be renewed.

Dr. Stowell reports two serious cases, with very high temperatures, 110° and 115° F., who were successfully treated at the Massachusetts General Hospital in 1892, by rubbing with ice. The detail of procedure, which was practically the same in both cases, may be profitably quoted.

A well-developed, muscular Scotchman of 32, assistant cook in a restaurant, after feeling dizzy a couple of days, left his basement kitchen at noon of June 14th, to walk off his vertigo, was seen to fall and was picked up unconscious, having a temperature of 110° F.; an ice-bag was placed on his head and he was taken to the hospital, where he was admitted with hot, dry skin, pupils moderately contracted, lips ashy pale, breath hot but sweet.

The second, a well-developed Irish laborer of 24, after working in a sewer, was seen to stagger and fall on the railroad track; he was given whiskey by a police officer and taken to the accident-room at 4:15 P.M., June 15th, completely unconscious, with a smell of whiskey in the breath; pupils much contracted and insensitive; skin hot and dry; lips ashy pale; pulse 146, very weak and irregular in force and rhythm; respiration superficial, irregular, labored; moaning and spasm with expiration; absence of knee-jerk; plantar reflex markedly excited by touching sole with ice; rectal temperature 115°, which was as high as the thermometer registered; was stripped on table and rubbed with ice, commencing on head, three and at times four men rubbing the whole surface except the præcordia; ice packed under neck and over carotids, and laid in the axillæ and groins; after twenty-five minutes rectal temperature had fallen to 104.6°, pulse still irregular and much weaker; atropine gr. $\frac{1}{60}$ given hypodermically; respiration somewhat deeper, moaning ceased for a time, goose-flesh began to develop and knee-jerk returned; moved legs and arms and resisted introduction of thermometer in rectum; ten minutes later, at 4:50 P.M., temperature 100°, pulse 132, character not improved; strychnine gr. $\frac{1}{60}$ given subcutaneously; pupils had dilated and now reacted to light, but he disliked to have eyes opened; goose-flesh had increased; bluish tinge to face and arms, latter folded across the chest in spasm; rubbing with ice stopped, except on head; at 5:15 P.M. temperature 98.3°, character of pulse unchanged; strychnine gr. $\frac{1}{60}$ given subcutaneously; removed to a dry table; moaning ceased for a few moments and then began again; dusky capillary circulation in cheeks and hands, did not return rapidly after pressure; respiration not deep or full; oxygen given in constant gentle stream and in ten minutes inspirations became deeper and expirations freer; legs partially drawn up on abdomen; head still rubbed occasionally with ice; skin of cheeks and shoulders warm to hand, of legs and abdomen cool; turned on side and drew himself together as if cold; opened eyes but did not understand; covered with blankets but moaning respiration and spasm at expiration returned in lessened degree; 5:45 P.M., temperature 96.6°, pulse soft, more regular, fuller and stronger; transferred to Ward D, feet and legs felt cold, face blue; oxygen as before for fifteen minutes, heater to feet, and blankets; ice bags to head and back of neck; pulse 128, constantly improving; 6 P.M. temperature in axilla 97.5°, quiet, opened eyes on being spoken to; 7:15 P.M., warm again; heaters and blankets taken away; delirious, fought violently to get up; rectal temperature 100.2°, head hot; put into tub-bath at 70° and held there in spite of struggles; head rubbed with ice which was also added to bath; in fifteen minutes began to get cold and goose-flesh reappeared; to bed again in moist sheet; 10 P.M., temperature 100°; pulse 80, full, soft, good strength; 11 P.M. talked rationally; respiration gentle; turned over and went to sleep. 16th, took ice-bags from head; 17th, discharged well.

In Dr. Burrall's cases at the Presbyterian Hospital, New York, the patients were placed on a Kibbee cot, in which they lay, wrapped in sheets wetted with water at about 65° F., supported on a netting with a trough placed beneath it; ice-water was poured over the surface of the body; ice applied to the head, mustard to præcordia and hot bottles to the feet; hypodermic injections of ♏xxx. of equal parts of whiskey and ether, of whiskey alone, or of whiskey and digitalis were administered as often as seemed necessary.

Dr. Hackley's practice at the New York Hospital was to rub the patient with ice or place him in a bath at 75° F., with ice to the head, and when the thermometer registered about 103° to remove him wrapped in a wet sheet to a fever cot, and on a further fall of temperature, to discontinue the pack and administer brandy per rectum or brandy and digitalin hypodermically, sprinkling the surface with ice-water from an ordinary watering-pot on further rise of temperature, and controlling convulsions by ether.

Illoway recommends in infantile cases that the naked child be placed in a folded sheet wet with cold water, laid in the cradle, and covered with a blanket, replacing the sheet as soon as warm, thus avoiding struggles on the part of the child to escape the bath and possible injurious shocks to the system from the latter, reducing the temperature more slowly, and with less probability of secondary elevation.

Five serious cases of the sthenic form of heat-stroke are reported by Dr. Haubold, house-surgeon of St. Vincent Hospital, New York, as having been successfully treated in the summer of 1890 by immersion in cold bath (60° F.) simultaneously with venesection to the amount of sixteen to twenty ounces. One of these was a blacksmith of forty-four with a rectal temperature of 107° F., one a laborer of thirty-seven with 108.5°, one a bricklayer of thirty with 109.5°, a laborer of thirty-two with 109.5°, and a coalheaver of fifty-four with 110.5°. All were comatose on admission, surface dusky, skin hot and dry, face flushed, pupils contracted, respiration stertorous, pulse full, bounding, 140 to 150; in the third, there was muscular rigidity amounting to opisthotonos, and in the fifth, the most critical, in whom the temperature was 110.5°, there was clonic spasm, simulating status epilepticus.

Flint states that on August 9th, 1862, eight cases of coma from sun-stroke were admitted into Bellevue Hospital, of which seven died. In the last case admitted, in which the coma was as profound and the symptoms generally as unfavorable as the others, the pupils were contracted, breathing stertorous, skin hot and dry, pulse full and frequent; bleeding was resorted to mainly because of the fatal termination of all the others, sixteen ounces being taken from the arm and the temples being freely leeched, and on the following day the patient was comfortable, complaining only of debility. In the summer of 1864 Flint had a similar aggravated case in which the abstraction of eighteen ounces of blood and the cold douche constituted the treatment. Consciousness returned in an hour and a half after venesection, and he was discharged the following day quite well. Nevertheless, the practice has few advocates in our own country and is generally condemned by British and East Indian practitioners, who with equal unanimity depend upon the cold douche. "While venesection may do good in some cases," says Wood, "in by far the majority of instances it does absolute harm." One of the exceptional cases in which it did undeniable good was that of Dr. S. Weir Mitchell, who having overheated himself by walking in the sun and making considerable muscular exertion, all of a hot August day, became unconscious after dining and remained so for ten days, being treated by cold applications, etc., finally recovering consciousness after a prolonged hot mustard foot-bath, and when able to speak insisted so positively upon being bled that twenty-five to thirty ounces of blood were abstracted, with immediate relief and speedy recovery, when meningitis and permanent mental derangement might otherwise have been the unfortunate and most regrettable result.

While the hydriatric treatment of hyperpyrexia is that now generally practised, other antipyretics have been recommended. Shattuck says that antipyrin has been given in a few cases in the Boston City Hospital with good results, and Dr. Westbrook of St. Mary's General Hospital, Brooklyn, reports two cases, one a robust young Englishman whose rectal temperature was 109° and the other an Italian laborer in whom it was 110°, both with symptoms of most profound vital disturbance, in whom the rapid and permanent reduction of temperature, he thinks, must have been mainly due to the hypodermic administration of a fifty per centum solution of antipyrin—inasmuch as the cold applications were only continued half an hour. In the first, he adds, "the beneficial effect of the abstraction of blood (thirteen ounces) was evident." In other hands large doses of antipyrin have been ineffective. In one of Katzenbach's patients, a large, heavily built female servant of fifty, five-grain capsules of acetanilid were the antipyretic used along with ice-cold cloths to the head, sponging with ice-water and vigorous fanning without intermission until consciousness returned and she protested against their further continuance.

Of 43 cases admitted into Bellevue College Hospital during the summer of 1868, of which 30 recovered, 1 of these having a rectal temperature of 110.5°, ice to the head, cold sponging, dry cups to the chest to relieve the pulmonary congestion, dyspnœa, and cyanosis, sinapisms to the extremities and stimulating enemata were the ordinary procedure. In 17, of whom 4 died, potassium bromide was freely used, and in 26, of whom 9 died, it was not used, the remedy consequently having been of no apparent benefit.

Ether in this country and chloroform in India have been generally used to control convulsions—the anæsthesia in cases of recovery having been maintained for several hours. Dr. McVicar, among a number of severe cases on board the steamship *Coptic* in the Red Sea, reports one of a steward, aged 30, who was found about noon unconscious from apoplectic coma, with pupils contracted to pin-points, injected conjunctivæ, pale face, rapid stertorous respiration, skin intensely burning and twitchings of the whole body; he was successfully treated by ice, sinapisms to nape of neck, inside of thighs, and soles, croton oil as a purgative derivative, hypodermic injections of ether to control the convulsions, and of pilocarpine gr. $\frac{1}{4}$ and atropine gr. $\frac{1}{100}$ to induce free sweating. Flint states that hypodermic doses of physostigma repeated two or three times at short intervals seemed to be useful in relieving muscular rigidity and convulsions. Lotions of ether, dry cups to the spine, and the rectal injection of ipecacuanha have been used in Australia to the same end. Hutchinson, on the strength of 16 cases at the Pennsylvania Hospital, of which 3 were immediately fatal and 1 after eight days, claims very great success following the subcutaneous injection of doses of one-quarter of a grain of morphine sulphate as a remedy for excessive jactitation, restlessness, and convulsions, ice and the rectal administration of brandy being also employed. Like success was had by Ellis with hypodermic injections of morphine sulphate gr. $\frac{1}{4}$, atropine gr. $\frac{1}{120}$, in overcoming the very violent convulsions, in one of his recent serious cases, a large, muscular sugar-house employé of forty-two, who after attempting to struggle against the sensation of "feeling badly all over," fell unconscious with a rectal temperature of 109° F.; further treatment was to deluge him with ice-water thrown from a height of four feet and to administer subcutaneously tincture of digitalis or tincture of strophanthus ♏x., along with whiskey ♏xx.

Confirmatory evidence of the value of morphine is given by Drs. Coplin and Bevan, who had an unusual opportunity of observing and treating heat-stroke in its onset in July, 1892, at the Franklin Sugar Refinery in Philadelphia. At the request of the management, an accident-room was fitted up by them, the temperature of which was cooled by an air-blast driven by an electric motor, and which was provided with a spray obtained from an artesian well furnishing water at 45° F. Of 213 cases occurring in eight days among 800 men employed by day and 500 at night, 182 were returned to work, 28 sent home, 2 to hospital, and but 1 died. Sixty had temperatures over 104°, the fatal case 110°. The routine treatment consisted of cold baths; friction with coarse rough sponges, to which they attach great importance as contributing to "increase the peripheral circulation," followed by application of a fine spray of water of a temperature of 45°-55° F., the forcible impact of which, practically amounting to flagellation, maintained the counter-irritation; and hypodermatic administration of $\frac{1}{20}$ grain of strychnine, $\frac{1}{40}$ grain of atropine, $\frac{1}{6}$ grain of morphine and 20 minims of tincture of digitalis, with drachm doses of aromatic spirit of ammonia by the mouth or morphine in combination with the ammonia was given by the mouth in doses not exceeding quarter of a grain. In mild cases antipyrin and antifebrin were beneficially used, with the ice-cap and cold spray to the head and nape of neck to relieve headache, cramps, and reduce temperature. In one case in which the rectal temperature was 103.6° after the usual treatment, including hypodermic injections of $\frac{1}{60}$ grain of atropine with $\frac{1}{6}$ grain of morphine, there was Cheyne-Stokes respiration, and the lips, ears and finger-nails became blue; the man was bled to eight ounces with immediate relief. Amyl nitrite was used in another on account of its promptness in inducing peripheral determination of blood, the headache and cramps disappearing almost magically, and the temperature falling in a few minutes from 102.6° to 99°. Nitroglycerin is suggested as an advantageous substitute for amyl nitrite. Subsequently to the active treatment, the patients were kept exposed to the cool-air blast, which Drs. Coplin and Bevan recommend as a most valuable adjunct, with active friction and rest in the recumbent posture.

There is nothing special in the after-treatment of convalescence from heat-stroke. Small doses of quinine are advisable; the combination of this remedy with aromatic sulphuric acid, as recommended by Wood, has been found by me, during an experience of many years in the Navy, the preferable form of administration.

Sequelæ.

Heat-exhaustion leaves no sequelæ, but after recovery from heat-stroke there are often lasting traces of injury to the nervous functions not limited to the vaso-motor and thermogenic. Persistent or frequently recurring headache, intolerance of light, chromatopsia, clouded vision, buzzing in the ears, deafness, vertigo, insomnia, loss of memory, mental weakness, alienation, irritability, altered disposition, tenderness over the spine, twitching of the muscles, epilepsy, cutaneous anæsthesia or hyperæsthesia, partial paralysis, abnormal condition of the reflexes, dyspepsia, obstipation, *diabète maigre*, anæmia, functional disturbances of respiration and of the heart's action, and many others have been enumerated by the various writers as enduring results of exposure to excessive heat. Permanent irritable condition of the heart, ending in organic disease, is so common among the engineers' force in the navy, that medical examiners for recruits without previous knowledge of their occupation recognize the "fireman's heart" among applicants for re-enlistment. It is the common experience in the navy that officers and men severely sun-struck or prostrated by heat are never afterward able to endure exposure to even moderately high temperatures, are subject to headache, tinnitus aurium, and other disturbances of innervation, are abnormally susceptible to stimulants, and become habitués of the sick list for apparently trifling complaints. Maclean refers to "incapacity for service in India or any hot climate without at least a more or less prolonged stay in a cold climate," as a consequence of heat-stroke in British tropical possessions.

The anatomical lesions on which these sequelæ of heat-stroke depend are, as might be inferred from the symptoms, chronic meningitis, encephalitis, myelitis, multiple and peripheral neuritis. To account for the ocular trouble, Alleman says, "It seems by far the most probable hypothesis to assume that this same disturbance of the nervous system which produces the general symptoms—such as nausea, vomiting, headache, giddiness, numbness, and convulsive movements—extends at the same time to the vaso-motor apparatus of the eye and then manifests itself as that nutritive disturbance which we recognize as papillitis. Visual defects, attributed by applicants for pension to remote attacks of heat-stroke, are very common, but in the opinion of Dr. Spalding, an experienced medical examiner, "insolation rarely affects the sight permanently and if so, the results in the form of optic atrophy ensue within a few months, never, as the innumerable pension claimants assert, at any interval extending from four to nineteen years later; and Dr. Tuttle, himself affected with night-blindness due to atrophy of the retina, consequent upon severe sun-stroke in June, 1863, while on picket-duty, agrees that if the disturbance of vision did not occur at the time or shortly after a man received a sun-stroke in the army, it is extremely improbable that blindness developed ten or twenty years after can be properly connected with his insolation.

Mental aberration eventuating in decided insanity is not an unusual consequence of the cerebral meningitis set up by heat-stroke. According to Pepper 3 of the 10 recoveries among the 20 cases treated at the Pennsylvania Hospital resulted in insanity. Wood agrees with Weir Mitchell that the serious after-lesion of thermic fever is meningitis, of which the fixed intense headache is symptomatic. Levick found extensive inflammation at the base of the brain in the case of a boy of seventeen, who had been sun-struck; and Mitchell relates the case of a former ordinary seaman in the navy, who had suffered from exposure to excessive heat on the coast of Africa, in whom he found extensive meningitis at the base of the brain, and another case of an officer in the army, sun-struck in Texas, who was treated for chronic inflammation of the meninges of the upper part of the cervical cord and of the base of the brain.

Eugène Dony quotes ("*De la Folie consecutive à l'Insolation*") the legendary account of the inhabitants of Abdera, who were struck mad after having assisted at a representation of the Andromache of Euripides exposed to the full sun, as evidence of the occurrence of mental derangement early in sun-stroke; and Battic (1758) reports the case of a sailor who suddenly became completely insane, from the sun darting its rays perpendicularly on his head, an instance akin to that told by Froissart of Charles VI., who became insane at the head of his army, from having remained on horseback all day in the sun during his march against the English. Didard makes the statement that Auvergnats, who go to the south of Spain, often return melancholic or otherwise demented. Bertrand, who was with Marshal Bugeaud's expedition in Arabia in August, 1836, stated that there were 11 suicides among about 200 cases of sun-stroke, and other French army surgeons noted similar occurrences at Oran and Philippeville. Fruchstersleben mentions sun-stroke as the cause of several cases of furious delirium. Dony further gives Pinel and Esquirol for authority as to the frequency with which mental derangement follows sun-stroke, the latter attributing 16 among 710 cases of insanity due to physical causes, to *coup de soleil;* and Dony himself, at the hospital at Bethlehem (1846–60), had 13 cases of insanity caused by sun-stroke, of whom 7 recovered. Decorse, while in Mexico, had under his care a pharmacien militaire and an army officer of rank attacked with sun-stroke, and two years later, at the Hôpital du Gros Caillou, he saw the pharmacien with paralytic dementia and learned that the other officer was under treatment in a private asylum; and at Charenton he had 3 patients affected with mental alienation after sun-stroke, 2 of these with melancholia, and 1 with decided mania, all three with a tendency to complete dementia. Vallon also reports two cases of general paralysis consequent upon sun-stroke.

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FROST-BITE.

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FROST-BITE:

CONGELATIO—AMBUSTIO EX FRIGORE.

THE vernacular expression *frost-bite* is legitimately applicable to the entire class of cases induced by low degrees of cold, from chapped hands and feet to the individual frozen to death (*asphyxia congelatorum*), as the parallel term heat-stroke is inclusive of all forms of lesion from excessive heat. Practically, however, frost-bite has come to have a limited application to more or less severe local effects and is treated exclusively as a surgical affection.

If it be desirable to have a distinctive designation for cases of general freezing, with constitutional involvement from the outset, loss of consciousness, torpor, coma, etc., *Congelatio* (*freezing*, *congélation*, *congelacion*, *congelazione*, *Erfrierung*) may be used as such, but even in cases of rapid general freezing there is a brief period of approach, indicated by local symptoms—the *frost-bite* of surgical treatises, *ambustio ex frigore*, *Frostbrand*, *gelure*, *quemado del hielo*, *geloni*—which terminates in death if the exposure be continued, or if the cause be removed and proper remedial measures be applied, is the limit of destructive action.

Less serious local effects of low degrees of cold are *Chilblains*, *kibes*, or *mules*—*pernio*, *frost erythema*, *erythema à frigore*, *Frostbeule*, *engelures*, *sabañones*, *pedignone;* or the still milder *chaps*, *fissuræ*, *Schrunde*, *gerçures*, *grietas*, which affect the lips, ears, fingers, and toes of persons with very sensitive skins.

Besides these are the phlegmasial consequences of "taking" or "catching cold," from a "stitch in the side" or a "cold in the head" to an acute catarrh or the gravest pneumonia or rheumatism, in which there may have been only a sudden fall of a few degrees of temperature in winter, or a quick cooling of the body from draughts, rain, or imprudent change of clothing in warmer seasons, and in which the acute inflammatory process developed is less the result of the mere comparatively lower temperature than of the constitutional predisposition of the individual or pre-existent morbid tendencies in the several organs or apparatus. The cold, in these instances, is appropriately considered among the exciting causes of the inflammatory or febrile condition set up, and is not of that intense degree which reduces the vitality of the exposed parts and causes general congelation, frost-bite, chilblains, or chapping.

Dr. Hartshorne, of Philadelphia, has described, under the term *cold-stroke*, a train of symptoms consequent upon exposure on a night in February, when the temperature had fallen 50 degrees (Fahrenheit), occurring in a boy of fourteen, who had rushed from bed, barefoot, and in his night-shirt, and stood at an open window, to which he was attracted by an alarm of fire. Becoming thoroughly chilled, he awoke in the morning, feeling generally indisposed, suffering with severe headache, drowsiness, and vomiting; the skin moderately hot; the pulse hard and accelerated. The next day there were vomiting, catharsis, incoherent delirium, closing of the teeth firmly against food and medicine, but opening of them to receive lumps of ice. There was no clonic spasm but a disposition to close the fingers upon the hands and to throw out the hands and feet with impatient suddenness. The skin became moist, the pulse variable but constantly accelerated, and during the night he died. Hartshorne mentions a similar case of a gentleman in London, who was observed to be ill on the street, having been exposed to a sudden fall of temperature, and was sent home in a cab, becoming delirious and dying in twenty-four hours; and quoting Larrey as to the serious effects of sudden great changes of temperature, he states his belief that the rapid fall in these cases produced molecular disorganization, as in the instance of plants suddenly blighted by frost.

M. Parrod of the Hospice des Enfants Assistés at Paris also applies the term *coup de froid* (cold-stroke) to the profound chilling (*refroidissement*) of children, especially the very young, who having been housed for several days in close, unventilated apartments are sent out-doors for hours, heavily wrapped and protected, and on their return are stripped, while sweating profusely, and laid in bed or cradle scantily covered; these awake during the night, restless, agitated, coughing as with croup, with flushed face, hot skin, and frequent pulse; or they are observed to be pale, hollow-eyed, and in an apparently syncopal state, the mouth opening as if from nausea, moaning incessantly and vomiting; others again awake with hot skin, cough, and broncho-pulmonary affections.

The primary effect of dry cold, like that of heat, is the general stimulation of the vital powers, the circulation and respiration are quickened, the surface becomes aglow, the step more elastic; there is mental exhilaration and a general feeling of *bien-aise*. This condition continues a longer or shorter time according to the natural vigor of the individual and the maintenance of his physical activity. If the thermogenic process be not commensurate with the demand upon it, heat will be gradually abstracted, painful burning and acutely pricking sensations are felt in the hands and feet, which seem heavy and swollen; the fingers and toes first, and then the higher joints gradually lose their flexibility and are benumbed. A general feeling of uneasiness and pain is experienced, soon giving way to torpor and drowsiness. The blood recedes from the surface and accumulates in the internal organs; the extremities become white, shrunken, and later mottled. The circulation slackens, innervation is at fault, the respiration labored; an irresistible desire to sleep overwhelms the patient, and unless active measures be employed he dies asphyxiated.

When no longer exposed to cold and reaction is induced, heat returns, usually transcending the normal, and congestion, inflammation, vesication, ulceration, sloughing, and gangrene are successively developed.

It is possible for very low degrees of temperature to kill all the tissues of a part as if charred by heat, but cold intense enough to produce this result will usually at the same time kill the individual by the depression of the nervous and vascular systems. The part frozen ordinarily looks well for a while, then becomes discolored, passing from light blue to purple, and ultimately black, evolving the gangrenous odor. When local death is a primary effect (*gangrène d'emblée*—Legouest), the part is characteristically dry and shrivelled. Some of Owen's cases of sudden gangrene at Quettah shrivelled up, becoming hard and parchment-like, others remaining more or less like healthy members and not sloughing. Larrey says in his account of the campaign in Poland, when what he terms "*gangrène de congélation*" was not uncommon, "in some it was limited to the surface of the skin of the toes and heels, and in others the mortification attacked the deeper tissues to a greater or less extent, some losing their toes and others the whole of the foot."

Less degrees of cold like moderate heat produce pleasurable sensations until the capillary circulation in the exposed parts is obstructed, when redness and swelling occur and pain is experienced, disappearing with the removal of the cause or continuing until inflammation is induced with effusion sufficient to form blisters.

Etiology.

Human beings resist cold as they do heat, but for a much longer period than in the latter instance because the more frequent and observable effects of cold are local; but the power of resistance varies with the health, vigor, and determination of the individual. The poorer classes enfeebled by intemperance and inanition, those whose occupations necessitate exposure to cold and wet, and who have the habit of sleeping in their wet boots, such as lake-sailors, boatmen, and oyster-dredgers, and others who have to sit or stand passive and unprotected from cold winds, as hackmen, car-drivers, conductors, gripmen, and locomotive engineers, are apt to suffer with frost-bites of the toes, heels, feet, fingers, hands, nose, ears, lips, and cheeks. The ability to endure cold increases after adolescence, and men past forty years of age succumb only under extreme conditions. Closely fitting gloves, tight shoes, elastic garters, sitting with the legs pendant in a cold room or vehicle, or any other cause interfering with the circulation predisposes women particularly to frost-bites and chilblains, and many of this sex with susceptible skins suffer at the slightest fall of temperature on the approach of winter, with fissured lips, chapped hands, agnails, and erythema of the feet and ears. In such instances, the knowledge of the liability suggests the precautions of dryness, especially of the feet, loose woollen underclothing, protection of the hands and ears, substantial heat-producing food, fat meats, etc., and brisk exercise as effectually preventive of the occurrence.

Incidentally it is interesting to note the effect of prolonged submersion in water of a much lower temperature than that of the body as illustrated by the account given by Gant (*British Medical Journal*, March 13th, 1880) of Captain Wells's swim of sixty hours at the Westminster Royal Aquarium, London, in a tank filled with bay-salt water of the specific gravity 1.027, and the mean temperature 81° F. (75–87°) at the end of which his oral temperature was 97.2° F., the surface warm, pulse 64, weak, irregular, small and compressible, respiration normal, and no conjunctival congestion. Drowsiness supervened, erythematous patches appeared on the lower parts of the thighs and red marks in the flexures of the knees and in that of the right knee a few small sloughy spots. There was no subsequent desquamation.

Arctic travelers testify to the exceedingly low degrees of persistent cold that can be borne, but rapid alternations from heat to cold, especially when accompanied with moisture and when the distal parts of the extremities, in which the circulation is less active, are insufficiently protected, are followed by frost-bite of severe character. Gant says that in his experience of Canadian winters a very much lower degree of dry than of wet cold can be endured. Instances are numerous of persons overtaken by snow-storms and overwhelmed for days under dry snow, whose lives have been saved. Tédenat mentions the case of a man, seventy years old, who had been buried twenty-four hours under snow, who was under his father's care, and who escaped with superficial gangrene of the toes, and he quotes from Pilhes the case of a blacksmith who had been similarly buried four days under snow, and from Reeve that of a woman who remained eight days under a snow-bank six feet deep, both of whom were taken out alive but subsequently died of gangrene. More remarkable both for the time of incarceration and the fortunate result is the case related by Fremmert of St. Petersburg of a peasant, who was caught in a furious snow-storm in the winter of 1850–51 and inclosed in his sledge by a firm crust of snow, who had some bread which he ate on the first day of his imprisonment, but was without other food until he was found at the end of the twelfth day, when he was still conscious, his face yellow, his body greatly emaciated and several of his toes frozen, and who recovered without serious consequences. The French army medical officers, who took part in the Russian campaign, agree that there was little suffering so long as the temperature, however low, was uniform and the air dry. Larrey states: "During the three or four extremely cold days preceding the battle of Eylau (February 7th to 8th, 1807), when the mercury stood from −10 to −15° Réaumur (9.5° to −2° F.) up to the second day after the battle not a soldier complained of frost-bite, notwithstanding they had passed three days and the greater part of the nights from the 5th to the 9th of February in the snow and during the sharpest frost. . . . On the night of the 9th to the 10th of February, the temperature rose to 3° to 5° R. (39° to 45° F.) and an abundant fall of sleet on the morning of the 10th was the precursory sign of the thaw, which occurred during the day, the temperature remaining at this point for several days. From that moment large numbers of the troops, both of the guard and of the line, began to complain of acute pains in the feet and of numbness, weight, and uncomfortable sensations of pricking and formication in the extremities. The feet began to swell and became dull in color; in some a bright red was observed near the base of the toes and on the dorsum of the foot; in others the toes had lost motion, sensibility, and heat, and were already black and dry, announcing the development of mortification." He says further: "All the physicians who have written of this mortification (*gangrène de congélation*) consider the cold alone as the determining cause; nevertheless, if we pay attention to the time of the explosion of this malady, its course, and the phenomena which accompany it, we can convince ourselves that the cold is only the predisposing cause." Owen says of his Indian cases, the men were all in a condition of great debility, with a low standard of vital power, caused no doubt by the fatigue of long marching and exposure to the rigorous climate, the opposite of that to which they had been accustomed all their lives. During the Crimean war, both the English and French troops especially suffered from frost-bite. Macleod states that on January 21st, 1855, when the thermometer indicated 5° F., 2,500 cases were admitted to the French ambulances, of whom 800 died from the effects of erysipelas, pyæmia, and gangrene; and Legouest, in his account of the congelations observed at Constantinople during the winter of 1854–55, says the gangrene, which was sought to be attributed to ergotism and scurvy, was really the result of frost-bite induced by cold and moisture in constitutions of defective vitality and feeble power of reaction.

Symptoms.

The reports of military surgeons of their experiences with armies operating during cold seasons and in frigid regions, those of the medical officers of Arctic and Antarctic exploring expeditions, and of our own army medical officers at outposts in the Northwest, offer so little variety in the sequence of pathological conditions that the recital of one is characteristic of nearly all. The following cases as related by Major William E. Waters, surgeon of the United States Army, when he was stationed at Fort Bridger in Utah Territory, during the winter of 1867–68, are typical of frost-bite as commonly seen in this country:

(1) A private, 36th U. S. Infantry, 24 years old, of feeble constitution, escaped from confinement in guard-house, 8 P.M., March 30th, by digging under the walls, and was arrested after six hours' absence and brought back, having travelled several miles in the snow. Soon after leaving the fort his feet were wet by wading across a stream. When seen at sick-call, both feet were congested, swollen, and painful. Both great toes and some of the others and the outer edges of the feet were pale and mottled. Complained of sharp burning and pricking sensation, as if parts had been exposed to a hot fire, and was unable to bear them on the ground in walking. In this case recovery was effected without vesication.

(2) A healthy temperate man of 30, on the morning of December 28th, started afoot to drive cattle to the post, a distance of fifteen miles, the ground being covered with snow six inches deep. Losing his way he wandered about all day, and as night came on and the weather grew colder, he experienced a very painful burning and at the same time numb sensation in his feet, which had been cold for some time. Fearing freezing, he took off his boots and socks and found his feet swollen, red, and painful, his heels and toes being pale and mottled. After rubbing them well with snow, he put on his boots and socks, and walked all night, reaching the point from which he had started early the following morning. His feet were then very painful and congested over most of the surface, except at the points where the circulation is most languid, which were pale and shrunken. He immersed

them in cold water with great relief. The following morning vesication commenced about the toes, heels, and ankle-joints. The vesicles, at first colorless, in a few hours began to turn red, and finally assumed a dark purple color. Ulceration of one of the heels occurred and one of the great toes became gangrenous, the line of demarcation being formed about the metatarso-phalangeal joint, ten days after the exposure to cold.

(3) Dr. D—— passed the fort in the latter part of December and when about fifty miles from the post lost his way and remained out for several days and nights with the mercury below zero. He was well clad and, aware of the danger of freezing, was careful to keep his toes in motion as much as possible while riding. Notwithstanding his precautions, when he reached a settlement both feet were frozen and the usual phenomena of congestion and vesication presented themselves, and on the outer edge of one foot, a space over an inch in width extending from the little toe as far back as the os calcis, sloughed deeply, leaving an indolent ulcer which granulated slowly.

(4) A German stage-driver of temperate habits started west from the fort with an empty sleigh and six horses on the morning of February 9th. The snow on the table-land between the fort and his station was three feet deep. He was all day going ten miles, and at night turned his horses adrift and started to walk, wandering about all night, and was found at noon next day, stupefied with cold and so fatigued as to be scarcely able to walk. He fell asleep, and at 2 P.M. his breathing was stertorous, pulse almost imperceptible, body pale and cold, presenting the appearace of fatal collapse. His feet and legs for several inches above the ankles were frozen stiff as boards. His feet were kept in ice-water until they became soft and reaction was finally established. Consciousness returned after twenty-four hours. The following day the feet and ankles were red, swollen, and quite sensitive to the touch; vesication appeared on the 12th; and on the 17th the skin of both feet and the vesicles assumed a dark color. Motion of the feet gave rise to intense pain. On the 22d, well-defined superficial sloughs developed over both ankles, terminating in gangrene, requiring amputation of both feet a month later, with ultimate recovery.

(5) A heavy, stout-framed man of 30, over six feet tall, in good health, started with another man at three o'clock in the afternoon of January 1st, to walk ten miles, in the midst of a severe snow-storm. They were soon lost but pressed onward until two o'clock the next morning. when they found a place somewhat protected, where they remained until daylight, pacing up and down on the ice to keep warm, when they undertook to retrace their steps. Early in the day, his companion became exhausted from cold and fatigue and finally froze to death. He experienced no great inconvenience from cold feet, but at night they became numb, all sensation left them, and they seemed like dead weights. On the third day, crossing a creek, the ice broke and he went into the water above his knees, which produced a warm and grateful sensation, and enabled him to walk with more ease. The snow was two feet deep and afforded some support to his legs. When about exhausted from cold, hunger, and fatigue he

reached a cabin, fell against the door, and when taken in was unable to articulate. His boots and socks were cut off; his feet were white, shrivelled, and frozen stiff, and ultimately became gangrenous, lines of demarcation being established on one foot about the tarso-metatarsal articulation and on the other as high up as the ankle. Amputations were performed and three months later the stumps were cicatrized.

Assistant Surgeon Shearer of the United States Army relates a case which is typical not only of severe frost-bite but of the power of resistance of a vigorous, determined man. The patient, who was admitted to the military hospital at Fort Wallace, Kansas, on November 22d, with both feet frozen, had been herding cattle on the Arkansas River, and, having lost all his ponies, started to walk seventy miles north to the Kansas Pacific Railroad. On the second day the temperature suddenly fell to −7° F., a northwest gale blowing. He kept on against the storm, and on the afternoon of the third day had the good fortune to meet and kill a stray buffalo. Almost dead with cold and thirst, he drank greedily of the blood of the animal. He threw away his rifle, cut up his blanket in pieces, which he tied around his feet and again pushed on. On the evening of the fourth day, he was discovered endeavoring to dig a hole in which to protect himself, in the sand along the railroad track, which he had struck twelve miles west of his objective point. Eighty head of stray cattle were frozen to death during the storm. When found, his nose, cheeks, ears, and hands were badly frozen, and life in the feet was irrevocably gone. Lines of demarcation soon established themselves high up over the left instep, including part of the heel, and just above the right tarsometatarsal articulation, leaving the heel intact. Violent erysipelas attacked the right stump on the fourth day, extending to the hip, but the patient ultimately recovered and resumed his occupation as a herder in Montana.

Frost-bites are of frequent occurrence during our furious "blizzards," or winter snow-storms, attended with winds of great velocity and low degrees of cold, as during that of February, 1895, when within a mile of the city of Washington, a party of farmers, breaking through a snow-drift over six feet deep, discovered the body of a man, frozen to death, standing upright and bowed a little forward as if breasting the storm; and the hospitals were full of cases of local freezing of varying degrees of severity. Frost-bite is a favorite subject with the authors of Paris theses and writers for medical journals, but the individual cases are much alike, and whether published in Stockholm, Rotterdam or Ghent, St. Petersburg, Cracow or Budapest, Vienna, Milan or Lausanne, Algiers, Constantinople or Calcutta, the story is only of a greater or less destruction of parts, usually of the feet, requiring amputation, in which the operator's interference is limited to assisting nature's spontaneous efforts to separate the dead from the living structures.

Morbid Anatomy.

Briefly stated, when a part is exposed to cold the cutaneous capillaries contract, the blood is driven out, and the part becomes pale, cold, and feels numb and heavy. If the exposure be brief, the capillaries dilate and fill with blood, reaction occurs exceeding the normal, the part becomes lively red in color, with a peculiar burning, pricking, tingling sensation, at first not disagreeable. Moderate cold of this degree has a pleasant stimulant effect upon the whole system, increasing the force and frequency of the circulation, with universal exhilaration, the systemic stimulation being quickly followed by general depression.

If the exposure be longer continued, the blood retreats farther from the surface, which becomes pale, whitish, and later of a stone-color; there is loss of sensibility and power of motion, and stiffness of the part; the sensation attending reaction is no longer pleasurable but apt to be decidedly painful from over-distention and congestion from the returning volume of blood; the skin becomes red, hot, swollen, and sensitive; and a general feeling of discomfort and pain followed by torpor affects the whole system.

If the cold continues still limited to the part, or is suddenly applied in an intense form but not severe enough to chill the entire body, the exposed part will be completely frozen, perfectly insensible, shrunken, cold, stiff, hard, even brittle, livid and mottled in color from coagulation of blood in the subcutaneous veins, the appearance being similar to that produced by the too long application of an ether spray to a part and due to the same condition of blood stasis. The stagnant corpuscles disintegrate, molecular death occurs, sloughing and mortification ensue, and a line of demarcation spontaneously establishes itself and marks the limit of possible restoration. At the same time the general system is oppressed as if under the influence of a powerful narcotic; drowsiness increases; and the individual keeps awake only by a strong effort of will or the vigorous interference of others.

A still longer exposure and a greater extent of surface involved induce the accumulation of blood in the large internal vessels and viscera; the brain is engorged, serous effusion takes place, and fatal stupor, coma, and asphyxia occur.

The danger from frost-bite is, therefore, in proportion to the degree of cold, the duration of exposure, the extent of surface exposed, and the physical condition and power of resistance of the individual. Alcoholism contributes greatly to a fatal result in comparatively mild frost-bite, from which persons of temperate habits would speedily recover. If a part has been only moderately chilled, the risk will ordinarily be from excessive reaction; if it has been completely frozen, it will be from sloughing; in general congelation it is from fatal coma and asphyxia. Lebastard, in a comprehensive study of the mechanism of death from cold, based on his experience as *médecin aide-major* of a column of French troops, which left Aumale for Boghar and was overwhelmed by a hurricane of icy rain and sleet, which arose suddenly on the mountains and caused the death of nineteen men of the Fourth Zouaves at Tléta des Douairs, arrived at the following conclusions: first, in sudden and progressive chilling, death is caused by cerebral anæmia; second, in slow and continued chilling, it is due to cerebral congestion; third, in sudden reheating it is the result of embolism from clots formed by carbonic acid set free; fourth, death after partial freezing is produced most often by congestion, sometimes by anæmia, both due to capillary embolism from clots formed in the frozen part. Fremmert in his statistical account of frost-bite as seen by him in the Obuchow Hospital at St. Petersburg during the decade 1867–77, published in Langenbeck's *Archives*, states that the greatest number of admissions was not always when the weather was coldest but when the changes were greatest, as during early frost or long-continued violent wet winds. The total number of cases treated in the hospital during the ten years was 494, of whom 456 were men and 38 women. The admissions during the year 1870, when the number was greatest, amounted to eleven per centum of all the cases occurring in St. Petersburg (about 700), and represented the average proportion of those requiring hospital treatment; of these 3 died. Of 402 frost-bites among men, of whom absolutely accurate histories were obtained, 112 occurred in passive conditions, while asleep on the ground or in sledges, wagons, or in rooms not heated; and of these men 62 were intoxicated, as were 98 of the 192, who were frozen while actively engaged, either walking or employed in out-door occupations, or were tramps, peddlers, etc. Of the 38 women 7 were frost-bitten while asleep on the snow or in cold vehicles, 4 of these intoxicated; and 11 were pedestrians, or working outdoors or street-walking, 6 of these being drunk. Only 212 of the 494 were warmly clad. In 333 cases the inferior extremities alone were affected; in 105 the superior only; in 38 both; in 12 the limbs and other parts; and in 6 the limbs escaped, the parts frost-bitten being the ears, nose, cheeks, prepuce and glans penis. The right foot was oftener frozen than the left, from the habit of allowing it to hang outside the sledge while driving. Spontaneous separation of fingers and toes usually occurred in sixteen to eighteen days, but in one instance was delayed six weeks. Two hundred and twenty-two operations were performed on 134 patients, 15 being major amputations of one of more limbs. The general health was unaffected in fifty-two per centum of the cases; in twenty-three per centum constitutional disturbances resulted, hospital gangrene occurred in seven per centum, erysipelas and phlegmon in six, traumatic and pyæmic fever in over eight, septicæmia, cerebral embolism, and tetanus in rather less than two. Of 42 deaths, 18 were from pyæmia and septicæmia, 7 from intestinal ulceration and chronic diarrhœa, 4 from cerebral embolism and apoplexy, and the rest from tetanus, chronic alcoholism, pneumonia, marasmus, etc.

Chilblain.—The minor local effects of cold described as chilblain (pernio), which are only secondary incidents of mild frost-bite, are fruitful subjects of contributions to the transactions of medical associations and to the pages of medical periodicals. They are common occurrences in young people, especially girls of lymphatic constitutions and inactive habits, and are a frequent result of the exposure of cold hands and feet to a hot fire, or of other sudden alternations of heat and cold. They usually begin as an eruption of violet or dull red patches, not defined in shape, slightly raised or tumefied, and attended with severe itching or burning, at the site of which scarcely perceptible vesicles and later decided bullæ form, which rupture and lead to ulceration of an indolent character. The characteristic ulcer of chilblain has the appearance of a smooth, superficial excavation with thick white edges and a peculiar viscid slimy discharge. When the skin is very thick, as over the heels, the ulceration occurs in the form of deep painful cracks or fissures known as *kibes.*

Gant defines chilblain as a local inflammation of the skin of an asthenic type, subject, perhaps, to regularly recurring attacks of congestion, and occurring in three degrees: (1) simple congestion attended by great itching, alternating with periods of extreme tenderness to external pressure; (2) vesication; (3) death or sloughing of the affected portion of the skin and perhaps of the subcutaneous cellular tissue, forming an ulcer of indolent character. Gross makes three varieties, the erythematous, the ulcerated, and the gangrenous. Dubrac describes four forms of *engelures,—érythémateuse, phlegmoneuse, phlycténoïde* or *ulcéreuse,* and *gangréneuse.* Dmochowski and others consider engelure and gelure (chilblain and frost-bite) as two distinct maladies; but Legouest, Vallette, and Tédenat regard l'engelure as a first degree of gelure; Hillairet and Gauchet describe it as the first degree of the action of cold upon the skin, being comparable to the rubefaction of a burn of the first degree; Ternier, Kaposi, and the French dermatologists speak of it as a dermatitis, resulting from frost-bite occurring among scrofulous subjects and those of other anæmic predispositions, in its ordinary type consisting of an infiltration of the derma in all or almost all its thickness, not well-defined in extent, slightly elevated above the surface, and surmounted by an exudation, which separates the epidermis, constituting minute phlyctenæ or decided bullæ.

Osler, defining Raynaud's disease as a vascular disorder, probably dependent upon vaso-motor influences, characterized by three grades of intensity: (*a*) local syncope, (*b*) local asphyxia, and (*c*) local or symmetrical gangrene, says of *local syncope:* "This condition is seen most frequently in the extremities, producing the condition known as dead fingers or dead toes, analogous to that produced by great cold," —and of *local asphyxia:* "Chilblains form the mildest grade of this condition, which usually follows the local syncope but may come on independently—oftenest affecting the fingers and toes, next the ears, and more rarely portions of the skin on the arms and legs. During an attack the fingers alone, sometimes the hands also, swell and become intensely congested. In the most extreme grade the fingers are perfectly livid and the capillary circulation is almost stagnant. The swelling causes stiffness and usually pain, not acute, but due to the tension and distention of the skin. Sometimes there is marked anæsthesia." The facts that attacks of this sort may recur for years and be brought on by the slightest exposure to cold, and that local or symmetrical gangrene follows the local asphxyia, establish its pathological identity with chilblain.

Chilblains are exceedingly apt to become chronic or recurrent, and often result in hypertrophic deformities especially of the hands, loss of nails, and other ungual dystrophies. Guérin describes numerous irregularities (*engelures anormales*) in form and seat of lesion, and says that it is interesting to note that the cold and atrophied extremities from infantile paralysis, syringomyelia, articular affections, etc., constitute favorite sites (*lieux de prédilection*) for chilblains by reason of the simultaneous extrinsic and intrinsic lowering of temperature.

Treatment.

The effect of prolonged extreme cold upon the animal tissues being to cause occlusion of the capillaries, interfering with the innervation and nutrition, and finally death of the part, the manifestly proper course of treatment is to attempt to bring about the gradual restoration of the circulation and through this the return of warmth, which can be best accomplished by gentle friction. If a part which has been frozen even partially is subjected to high external temperature, before the circulation is restored, its death will inevitably result. Parts severely frost-bitten, or actually frozen, are not always irrevocably destroyed. As stated by Gant, the combs of cocks and the ears of rabbits were frozen by Hunter and these parts afterward recovered. Leeches and frogs have been placed in the same condition and afterward restored to activity, the duration of the congelation being tolerated longer in cold-blooded animals than in warm.

The ordinary practice is to rub the frozen parts with ice or snow precisely as in cases of heat-stroke, a procedure apparently as paradoxical as that of "blowing hot and blowing cold." No matter how cold, white, hard, and brittle the part, even should a whole limb or several be involved, if the freezing have not been too long continued, partial recovery may be expected by judicious and very gradual attempts to restore the circulation. The case related by Dr. J. T. Hayes of the Kane Arctic expedition is pertinent:

> "An Esquimau, who had his leg frozen above the knee-joint, until it was stiff, cold, and apparently lifeless, was placed in a snow-house having a temperature of −20° F. The leg was bathed with ice-cold water for about two hours and then enveloped in furs for three or four hours, when frictions were begun, first with the feathery side of a bird-skin, then with snow, alternately wrapping the limb in furs and rubbing it for nearly twenty-four hours. It was next carefully wrapped up and the temperature of the snow-house gradually raised by lamps above zero. On the third day, the patient was taken to his own house, and in seventy hours he was walking about with only a slight frost-bite of one of the toes."

Thorburn says it is difficult to determine how long torpor may exist without destroying vitality. Sir John Franklin declared that an animal could be resuscitated even after the whole body had been frozen. Ashhurst quotes Nicolaysen's report of a case of recovery after the rectal temperature had fallen to 76.4° F.

The precise measures to be adopted must be left to the discretion of the medical attendant, upon whose skill and ingenuity will depend the adaptation of available means to the peculiar exigencies of each case. Ice, snow, iced water, and cold water gradually raised in temperature, are the main dependences. Sometimes ice and snow may be too cold or rough, and cold water applied by soft sponges or flannels or by irrigation, sprinkling or spraying will be the better means. Sometimes friction is not justifiable even with the utmost care and gentleness, as when the frost-bitten parts are very brittle, as in the case of the external ears and tip of the nose, which have been broken off by injudicious manipulation. In profound general congelation, artificial respiration, persisted in for hours, will probably be required along with the local treatment. The febrile reaction after protracted exposure to cold is ordinarily of the typhoid grade, and brandy, whiskey, and other general stimulants by mouth or rectum will be indicated. Later in the case, slightly stimulating lotions of camphor, linimentum saponis, ammoniated embrocations, tincture of arnica, hamamelis, and the like may be appropriately used to encourage local reaction, which must not be allowed to become excessive, nor should the patient be placed in a warm room, nor be brought near a fire, until the normal circulation has been re-established; imprudences of this sort have resulted in fatal congestion of the lungs as well as in death of the part. According to Sir Astley Cooper, even the warmth of the bed may occasion inflammation liable to end in gangrene. Baron Larrey, writing of the expedition of Moscow, says: "Woe to the man benumbed with cold, whose animal functions were almost suspended and whose external sensibility was lost, if he suddenly entered into a room too warm or approached too near the great fire of a bivouac! The benumbed parts or frozen extremities, distant from the centre of circulation, were struck with gangrene, which manifested itself at the very instant and developed with such rapidity that its progress was perceptible to the eye. Or else the individual was suddenly suffocated by a sort of turgescence, which seemed to seize upon the pulmonary and cerebral systems; he died as from asphyxia. So died M. Sureau, *pharmacien en chef de la garde.* He arrived safely at Kowno only weakened by cold and abstinence. They offered him a refuge in a very warm room of the pharmacy of the hospital. Scarcely had he passed a few hours in this atmosphere, to which he was unaccustomed, when his limbs, which he no longer felt, became swollen and bloated, and he expired soon after in the arms of his son and one of his colleagues, without having been able to utter a single word." In another place, he says: "Persons have been seen to fall stone-dead at the bivouac fires. All who approached near enough to warm their frozen feet and hands were attacked with gangrene at all the points where cold had destroyed their vital properties. . . . Happily most followed the advice of the surgeons, rubbing with snow and with lotions of camphorated brandy, which prevented gangrene, when it was not already developed, while this occurred in all who went into the cities or near the fire." Of eight cases under the care of Surgeon Owen of the Indian service in the base-hospital at Quettah, occurring among camp-followers at the encampment of Dasht, six thousand feet above the sea level, five who hastened to warm their cold feet by the fires suffered with suddenly developed gangrene.

When local inflammation, which is usually disposed to assume an erysipelatous character, does occur, it must be controlled by astringent and refrigerant applications aided by position. Von Bergmann advises suspension of the frost-bitten member in an elevated position and Fremmert relied principally on vertical suspension and antiseptic applications. The list of local remedies recommended is almost interminable. The tincture and compound tincture of iodine have met with most general favor. The tinctura ferri chloridi, the application of lint soaked in ox-gall as recommended by ex-Surgeon-General Moore of the United States Army, aqua ammoniæ, tincture of arnica, castor oil, the various balsams, with hydrocyanic acid, etc., have had their advocates in the treatment of superficial frost-bite. Besnier and Broq in the *Lyon Médical* for January, 1895, advise bathing the frost-bitten hands in a decoction of walnut leaves and, after drying, rubbing them with strong tincture of camphor and then dusting with a powder of bismuth salicylate and starch, allaying the itching at night by a lotion of tannin, rosewater, and glycerin, followed by the use of the powder. If ulceration have occurred, the parts should be wrapped in walnut leaves softened in hot water. Acetanilid, iodoform, resorcin, salol, and the unofficinal ammonium, potassium, and sodium sulpho-ichthyolates have also been used.

Dr. Hermance reports the results of different remedial measures employed at the Polyclinic Hospital at Philadelphia, during the exceedingly cold period of the blizzard of the early part of February, 1895. Of 30 cases selected at random among the large number crowding the hospital, 5 applied for treatment immediately upon finding the parts benumbed, and these were subjected to prolonged friction with snow followed by douching with ice-water for five to ten minutes, and after this by the application of an ointment containing twenty-five per centum of ichthyol. Of the 25 who did not apply for relief until the parts had become hot and painful, 18 were treated with an ointment containing one-third its weight of ichthyol, 4 with benzoated collodion, and 3 were painted with a ten-per-centum solution of silver nitrate. Twelve who had been treated with ichthyol reported immediate relief from pain, 8 exhibited blebs, which were punctured and the ointment reapplied. Eleven treated with ichthyol required a second dressing, and in only 3 of these was any further attention deemed necessary. Two of the 3 painted with the silver nitrate solution were discharged cured; 2 of the 4 treated with benzoated collodion reported increase of pain and blebs had formed; the other 2 had not been benefited. Of the total 30, 9 were of a serious character, in which there were lesions of the entire skin and in some cases of the superficial fasciæ. These were all treated with ichthyol, until the parts became raw and exposed, when acetanilid ointment (ten per centum) was applied. The practical conclusions drawn from these facts were: first, that of the three drugs, ichthyol gave most relief from pain and satisfactory protection of the part; second, that when ulceration occurred, acetanilid ointment best prevented suppuration and promoted quick granulation and cicatrization; third, that in cases of great destruction of tissue and serious disturbance of the vaso-motor mechanism, as was the condition in 5 of the 30 under consideration, no treatment avails, and nothing can be done but wait until nature throws off the destroyed tissues, assisting in the mean time to keep the parts clean and inoffensive.

When sloughing begins, the customary treatment of this condition from other causes—by warm-water dressings, hot fomentations, emollient and stimulating poultices (the yeast poultice being a favorite of the older surgeons), and epithemata of spongio-piline, jute, or tow, impregnated with carbolic acid, glycerin, alcohol, turpentine, castor oil, etc.—is all that is necessary. Resin and tar ointment, ichthyol, carbolized oil, lime-water and linseed oil, or lime-water and cod-liver oil relieve the pain, promote the separation of the sloughs, and encourage granulation and cicatrization of the resulting ulcer. A case at Roosevelt Hospital, New York, reported in the *Medical Record* of October 10th, 1873, in which the parts over the patella had been frozen, sloughed and left an opening into the joint, was successfully treated by compression by hot sponges. Even moderate frost-bite usually ends in some death of the skin, shown by the blistering of the surface and according to Cheever by the occurrence of subacute eczema, especially of the ears, which are apt to be affected with eczematous inflammation for weeks. In other cases, the skin of the fingers peels off, the nails loosen and finally drop out.

When the parts are irrevocably dead, they must be removed, but surgical interference is rarely required, especially when the frost-bitten member is of minor importance and the sphacelus of small extent, until lines of demarcation have spontaneously marked the limit of destruction. The adroitness of the surgeon will be exhibited in preventing unnecessary mutilation, of which the inevitable extent is usually indicated by nature's own efforts to rid itself of the dead tissue. Grant says: "We have in Canada frequent and forcible illustrations of the unaided amputating powers of nature in the cases of such frost-bitten persons as remain for weeks in the 'lumber shanties' prior to being removed to a hospital." Much of the literature of frost-bite consists of reports of dexterous operations, especially about the feet, by which useful stumps have been obtained. When a whole hand or foot is unmistakably dead, it is better to remove the sphacelated mass, especially if offensive, by cutting through the dead tissue below the line of separation, waiting to improve the shape of the stump by a subsequent operation, when the patient's condition has been meliorated by proper constitutional treatment. The success of an operation depends on the general condition of the patient, and in civil life, particularly in well-appointed hospitals, the results are almost uniformly satisfactory. In the Crimea, the mortality as stated by Baudens was great because of the wasted condition of the sick, many of whom were affected with diarrhœa and other debilitating camp diseases; and during military operations generally, when frost-bite is frequent, the circumstances are not conducive to recovery.

The treatment of the mild form of frost-bite known as chilblain has nothing special. Rubbing with snow or immersion in cold water on the first appearance of the erythema, and afterward the application of iodine, silver nitrate, benzoin, turpentine, or other stimulating embrocations are ordinarily sufficient. In my own hands repeated use of strong solutions of carbolic acid has been most serviceable. The old-fashioned domestic remedy, brandy and salt, is extolled by Gant, and like many similar simples of our grandmother's pharmacopœias is, doubtless, quite as efficacious as the new-fangled compounds of the rival drug firms of the day, which replace each other and become obsolete before they are well established. Thorburn mentions copper sulphate and Fergus sulphurous acid as effective. Ashhurst recommends frequent painting with a weak (one per centum) solution of silver nitrate and then wrapping the part in raw cotton, which by obtunding sensibility relieves the distressingly painful burning. Lead lotions and opiate ointments have likewise been used to subdue intolerable itching. Periodical paroxysmal pruritus may be anticipated by the revulsive effect of immersion in a mustard bath. Gross says that in obstinate chilblains he has obtained great benefit from the application of a blister until thorough vesication is produced. Vesicated chilblains may be protected by flexible collodion and castor-oil varnish. After ulceration occurs, the sores are to be treated as other intractable weak and indolent ulcers, using mild citrine ointment, resin cerate, tar ointment, ichthyol, etc., as occasion indicates. When sloughing occurs, the act of separation is to be assisted as ordinarily by cataplasms and granulation encouraged by ichthyol. I have found the application of copaiba very satisfactory.

As before stated, persons once affected with chilblains of the toes, fingers, ears, nose, etc., are apt to have them recur and should protect themselves by proper clothing, by avoiding fires after being chilled, and by using stimulating lotions, such as dilute solutions of carbolic acid, by which I have prevented recurrence in several inveterate subjects.

The most serious constitutional sequelæ of severe frost-bite are affections of the nervous system. Peripheral paralysis is a frequent consequence, the nerves most affected being the radial and the facial. Larrey mentions that many of the survivors of the retreat from Moscow became hemiplegic.

Touraine and other French military surgeons describe cases of syncope preceded by excessive redness of the whole body as the result of cold bathing, similar to the effect in certain individuals, who after a decidedly cold bath are affected with a peculiar sensation in the entire periphery, as if produced by myriads of red-hot needles piercing the skin, which is relieved by pressure but reappears in distant sites, persisting until reaction has been established.

MacCollum relates a case of acute tetanus after frost-bite ending fatally on the seventh day; and McLaughlin one of recovery from gangrene with pyæmia after double amputation. Van Vyfe had a case of anasarca following exposure to extreme cold, and Thorburn mentions among the sequelæ congestion and inflammation of the lungs and ulceration of the duodenum, the latter a result also of intense heat producing burns.

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THE
DISEASES OF OCCUPATIONS.

BY

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THE DISEASES OF OCCUPATIONS.

Historical Note.

THE founder of this important branch of modern scientific medicine was undoubtedly Bernardino Ramazzini, a professor at Padua. In 1700 he wrote a Latin treatise, "*De Morbis Artificum Diatriba*," which was the first systematic work on the subject. This book must have attracted marked notice, for it was soon translated into English, and later into German and French. It is still worth reading, both for the individuality of its style and for the observations and quotations with which it abounds. It has, however, many faults, some of which are characteristic of its age and country, while others seem to be inherent in the subject itself, for they still mark the productions of some recent writers who probably would not acknowledge Ramazzini as their model. Thus the book is desultory and digressive to the last degree; the author does not scruple to wander off through several pages to discuss subjects foreign to his theme, as when for instance he takes eight pages to tell us that the cleaners of "jakes" (privy-wells) have sore eyes. He is painfully pedantic, and his quotations are not always to the point. He is, moreover, curiously superstitious, as when he tells us gravely that ghosts abound in mines to plague and terrify the miners. Finally, he is credulous to the last extent in his mediæval therapeutics. But such faults are venial, and delightfully characteristic of a learned Italian professor of the seventeenth century. His more serious faults include a tendency to generalize and to ascribe all the ill-health of an artisan to his occupation. In this he is still followed by too many writers. Thus in his chapter on chemists he describes affections that are not due to the practice of chemistry; again, he tells us gravely that millers are likely to be lousy; and he quotes Juvenal to prove that blacksmiths have blear eyes because Demosthenes' father was a blacksmith and had blear eyes. His descriptions of symptoms are weak, and his scientific knowledge is often defective. With all his faults, however, Ramazzini is to be commemorated as the first physician who saw this great subject as a whole and attempted to treat it in a systematic way. In his modest preface, while claiming justly that his treatise has in it something new and original, he states his belief that what he published was only "an incitement to others to lend their helping hands, till an intire and compleat Treatise be obtained, that may deserve a place in the Commonwealth of Physick."

The next writer of importance in this field was the illustrious Tissot. He wrote an essay in 1768 on the "Diseases incident to Literary and Sedentary Persons," which was translated into English in 1769. This essay is elegant in style but not always convincing in matter, because it has the still prevailing fault of ascribing too much to the alleged cause. In the cases of Tissot's scholars and literati, every ache, every symptom is to be ascribed to their occupation. The stone in the bladder is with him a concretion of learning. A most instructive case of hysterical sleep in a young man, lasting for a year, is an evidence of nothing but overstudy. The most notable part of his book is the description of neurasthenia (called by other names) due to mental overwork. He describes especially well the neurasthenic mental state with its imperative conceptions, inhibitory ideas, and strange delusions, and illustrates the theme from many sources.

Tissot refers, rather disparagingly, to two works that had immediately preceded his own. One, by Platner, had touched upon this subject only incidentally, and the other, by Pujati, was nothing more than a compilation of general dietetic rules. Neither of these authors made a permanent impression upon this department of medical hygiene.

Patissier's book was the next to appear. It was not, however, until 1822 that this author ventured into what was still an unfamiliar field. His book is founded upon that of Ramazzini. It is really a copiously annotated addition of Ramazzini, whose original text is used and inclosed in brackets. Much is added that is original and modern. As an example of Patissier's method and acumen the following may serve. "Pastry cooks," he quotes, "are less debauched than bakers, and more gentle and more sociable." He thinks he sees in pie-dough a mysterious influence that renders a man more sweet and companionable than his fellow who makes bread. This extravagant induction is on a par with much that has been, and is still being, written on the influence of occupation on health.

In 1831 appeared the first English work on the effects of arts, trades, and professions on health and longevity. This book, by C. Turner Thackrah, Esq., was in many ways creditable to the author and his country. It is rather remarkable, however, that England, which has been so conspicuous as a manufacturing nation, should have been so late in making her first notable contribution to this literature. Thackrah's book is commendable for the close personal observation that its author had given to the subjects that he had taken in hand. It is not free, however, from the fault that ever marks the early, and even some of the later, writers on these diseases. Too much is ascribed to the influence of occupation, and not enough care is taken to discriminate in favor of the innumerable effects upon health of defective hygiene, of vice and immorality, of bad heredity, and of the various infectious and constitutional diseases. The author takes up the various trades and occupations in turn and draws conclusions about them which are often both doubtful and unimportant. Thus he says that hostlers have congestions of the vessels of the abdomen and head which are due to their habits of drinking ale and spirits; soap-boilers are generally temperate; and comb-makers are healthy and long-lived. Thackrah's book is thus rather too much of a catalogue of trades, with commonplace observations. The drinking habits of hostlers are not distinctive nor necessarily inseparable from their occupation; some soap-boilers are probably not so temperate as others; and that comb-makers are long lived is too generalized a statement to permit us to learn from it much about the details of the effects, if any, of that occupation upon the health of the artisan. We call attention to these weak points in Thackrah's book because they characterize too much of the writing on this subject.

After the appearance of Thackrah's book the literature of this whole subject became rapidly more voluminous. This increase was due to two causes: first, to the rapid and extensive increase in all the manufacturing arts; and, second, to increased activity in medical and scientific research in all directions. As a consequence of this stimulation this department alone of medicine and hygiene now supplies a literature which it is quite beyond the power of the most laborious student to read and digest within the space of time allotted to the preparation of a condensed paper on these subjects.

For the purposes of this historical note it will be sufficient and only possible to refer to some of the most conspicuous and useful works. The object, moreover, of such an historical note can thus be attained; which is, to indicate along what lines of development this study has been most profitably pursued by the numerous contributors to it. From the crude work of Ramazzini to the statistical and scientific method of Hirt, of Layet, and of Ogle is a regular progression which supplies motives and hints for those who seek to still further elucidate the subject and to facilitate its study.

French writers have been especially prolific in books and articles on the hygiene of the professions. The three most eminent have been Lévy, Tardieu, and Layet.

In his treatise on hygiene, issued in 1844, Lévy devoted a lengthy chapter to the hygiene of occupations. In this chapter we find a condensed description of many of the diseases incident to tradesmen because of their occupations. This author was apparently the first to draw a distinction between the evil effects of a mechanic's vices and those of his trade. "It is unjust," he says, "to stigmatize the trades, as Cadet-Gassicourt has done, by an enumeration of the vices and the bad traits which are observed in those who practise them; it is not proved that their corruption surpasses that of other classes of society." This important distinction is observed by Lévy with more care and consistency than by some more recent writers who might have profited by his example. He wrote at a time when this whole field had not yet received the labors which in this day have resulted in such an elaborate literature; consequently his treatise is no longer complete for reference. It is nevertheless a model of method and construction, and therefore is still a work of value; and it is replete with shrewd and wise sayings. Recalling the fact that apoplexy had had illustrious victims in Petrarch, Copernicus, Malpighi, Richardson, Linnæus, Marmontel, Spallanzani, Corvisart, Walter Scott, and other savants and literary men, he says that these great names strike the attention, but they do not touch upon the question of the comparative frequency of apoplexy in the illiterate classes and in the class devoted to intellectual toil. In other words, the apoplexy of these great men may not have been caused by their pursuits. When one recalls, in fact, that Sir Walter Scott led largely the outdoor life of a country squire, and, on the other hand, that Richardson was a close London shopkeeper, it is seen at once how absurd it is to associate these two together as likely to have suffered from the same disease simply because they both wrote novels!

Tardieu's well-known dictionary of hygiene appeared in 1852. It is a mine of information on all departments of hygiene, including occupation, up to the time of its publication.

Layet's treatise on "Hygiène des Professions et des Industries" is the most recent and on the whole the most useful French work on this subject. It is thoroughly scientific and gives constant evidence of the direct personal acquaintance of the author with his great variety of topics.

In addition to these eminent authors the French have had, now for many years, the *Annales d'Hygiène Publique et de Médecine Légale.* This journal has been the depository of much of the most valuable literature relating to our present and kindred topics. It is a mirror, in fact, in which is reflected the progress of all the hygienic sciences for more than a half century.

In Germany the representative book is Hirt's "Die Krankheiten der Arbeiter" which appeared in 1871. It is rich in statistics and bibliography, and is the work evidently of a close student of trade conditions and tradesmen as presented to the author's observation in Germany. On the subject of his statistics, and on statistics in general, more will be said in a special discussion which is reserved for a future page. Hirt's work is a standard, and will remain such. Germany has produced an immense number of other contributors to this subject, either in monographs or in journal articles, but they cannot be mentioned individually.

The distinct tendency in England in recent years has been to treat this subject from the standpoint of the sanitarian and statistician. Scattered through the voluminous reports of the Local Government Board, of the Registrar General, and of the Privy Council, may be found an immense mass of material bearing upon these aspects of the subject. For the purposes of this article very much of this material cannot be utilized. However valuable it may be in itself, it becomes obvious at once to any one who examines it that it occupies too vast and generalized a field for the purposes of a study of the distinct and special diseases that are directly caused by occupation. Thus the sanitarian approaches the subject for the purpose of throwing all possible light upon the conditions and environments of the various trades and divisions of labor. He includes much that belongs properly to the subject of hygiene, such as locality, diet, habitation, moral and physical conditions, heredity, and the prevalence of the various epidemic and constitutional diseases. A description of all these conditions, which of course affect the health of the laborer, does not include necessarily, or, if it does include, does not sufficiently indicate, the special affections that are the direct products of trade-processes. The deductions drawn from these studies, moreover, are not always above doubt. For instance, taking the case of the cotton spinners, it is a task of the nicest discrimination for the sanitarian to determine how much of their health may depend upon their occupation; and how much upon the conditions amidst which they live, which latter must vary according to country, to locality, to race, to habits, and even to the state of the market.

The same criticism applies even more pertinently to the work of the statistician. The prevalence of any given disease, as for instance phthisis, among any section of artisans, must depend, as in the bulk of the population, upon many causes outside of their special occupation; and their mortality rate, as made up in comparative tables from the death returns, affords, it seems to me, a very doubtful light upon the effect of their particular occupation upon their mortality from this or any given affection.

Among those who have done notable work in these fields may be mentioned Sir John Simon and Dr. Headlam Greenhow, in the Medical Department of the Local Government Board; and Dr. William Farr, the eminent statistician, whose use of the returns of the English Registrar-General's office has given him great fame, and undoubtedly conferred benefit upon his country and mankind so far as this method is sound and reliable.

Very searching and valuable studies of insurance data and statistics have been made also by experts, but they cannot be summarized here. These labors do not throw an especially strong light upon the subject under discussion.

Among recent contributions to the special subject of the diseases of occupations are the admirable paper by Dr. Roger S. Tracy in Buck's "Treatise on Hygiene"; and the chapter on the same subject by Dr. Hime in Stevenson and Murphy's "System of Hygiene." The most recent work, and the last that I shall mention in this brief note, is Dr. J. T. Arlidge's valuable treatise on "The Hygiene, Diseases, and Mortality of Occupations," published in 1892. It is noteworthy from the title of Arlidge's book that it is not confined strictly to the "diseases" of occupations but takes the broader field of "hygiene" and "mortality." The fact serves to exemplify the tendency, above referred to, of most recent writers to enter upon a broader field than that strictly of consequential disease. This tendency is directly due apparently to the extraordinary, even inordinate, development of the statistical method not only in England but also in France and Germany. In England, as already said, under the influence of such sanitarians as Simon and Neison, and such statisticians as Farr and Ogle, the subject has been made to include many cognate and correlated topics and has been subjected rigorously to the dominion of statistics. In this way the distinct and special subject of the true trade-diseases, *i.e.*, the diseases that are caused by occupation, has been in a measure lost sight of in an attempt to draw broad conclusions about trade-hygiene, trade-incidents, trade-mortality, and trade-legislation. These subjects are not necessarily associated intimately the one with the other. For instance, trade-mortality, *i.e.*, the rate of mortality from all causes among men working at a certain trade, may depend to only a slight extent upon the diseases that are directly due to the trade. This is so because artisans, like other men, die from all kinds of diseases. Arlidge's book shows very clearly the effects of this statistical and sanitarian method of study, although he himself makes but a limited use of statistics, except those that he borrows. Hence it is not a little overloaded, for our purposes, with extraneous and even superfluous matter, and has the fault of a rather diffuse style. For the English reader, however, it is the most useful book extant.

The great proportions to which the literature of this subject has grown are nowhere better shown than in the index-catalogue of the library of the Surgeon-General's office, U. S. A., in which, under the heading of "Occupations and Trades," and of numerous cross references, many pages are given of titles of special monographs and journal articles on all varieties of these topics. In the presence of such a bibliographical storehouse it is fortunately superfluous for authors to overload their pages with references.

CONDITIONS OF OCCUPATION WITH REFERENCE TO THE PREVALENCE OF DISEASE.

The student, when he enters upon an inquiry into the diseases of occupations, is met at once by the necessity for defining the subject. What is to be understood as a "disease of occupation"? Obviously it will not do to include in the term every disease affecting the artisan. Neither, if the term is strictly guarded, can it be allowed to embrace every disease that is affected by occupation. For, in the first instance, as artisans have the ordinary diseases of humanity, to include all their diseases in those "of occupations" would be to destroy all distinctiveness in that term. But, in the second instance, the latitude implied is not easily defined, for it is an undoubted fact that some occupations tend to promote certain general diseases, as mining causes rheumatism and as silk-weaving is popularly supposed in Lyons to favor the spread of tuberculosis. Such diseases may properly, under such circumstances, be claimed as coming strictly under the heading of "diseases of occupations"; that is, of diseases caused or promoted by the trade or business of their victim. When, however, this matter is considered for a moment it is seen how widely and indeterminately this whole subject is thus spread abroad, if account must be taken of every disease that may, however remotely, be influenced by occupation. The special and distinct meaning, which is naturally attached to the term, is at once destroyed by such a liberal interpretation. In the cases of general, constitutional, and even infectious diseases the predisposing causes are usually exceedingly difficult to discover; they may exist in errors of diet, of habitation, of personal hygiene, as well as in the occupation. To distinguish these causes is the most difficult task of the sanitarian, aided by the statistician.

The scope of this paper is limited to a description of those injurious effects or diseases that are direct, characteristic, and indisputable. Thus the lead-poisoning of a file-maker, the mercurial poisoning of a gilder, the anthrax of a wool-sorter, the paralysis of a penman, and the paraplegia of a laborer in a caisson, are directly due to the occupation, are characteristic of the effects of the occupation, and are indisputably caused by it; without the occupation they would not have occurred to the individual concerned; and with the occupation there was beforehand a very good chance that they would so occur. On the other hand the bronchial catarrh of a street-car driver, the anæmia of many ill-paid artisans, which is caused rather by poor food and bad lodgings than by the pursuit of their trade, or the tuberculosis of silk-weavers, will not be described. To describe all such affections, which may be due to other causes, such as poor hygiene, vicious habits, infectious processes, and even heredity, and to strain a point to ascribe them to the various trades, would stretch this paper beyond all reasonable limits. Yet there is a temptation to pursue this plan, and it has been pursued by not a few writers. Every trade is taken up by them in turn, and its supposed ill effects are described, allowance not always being made for the innumerable other causes that may, and constantly do, affect various bodies of men who are aggregated for purposes of labor. The obvious facts even that locality, race, age, customs, habits, and country affect artisans quite as much, even more, than do their occupations, are too readily ignored; thus the butchers of one race or town may be more healthy than those of another, although following identical occupations; or, as Arlidge states, the cotton-spinners of one mill may have a great advantage over those of another because they happen to work upon a special fabric for which there is greater demand in the market, hence receiving steadier wages and leading healthier and more secure lives. It can readily be seen to what vast proportions the subject extends, and how unfortunately its details may become confused, if some such rule as that which I have adopted is not observed.

The value of statistics in a study of the health of a laboring population is not to be doubted. The only question is, What is it desired to show? Statistics can do some things, but not everything. They supply a method, but they are not, as some have claimed, a science in themselves. If properly compiled, and guarded with control-tables, they may give important information. The value of statistics may be said to depend entirely upon the value of their primary factors. Thus, if the primary factors or units which compose a statistical table are of known value in themselves, or even of approximately known value, then the statistical inductions drawn from them are of corresponding value. If, however, the primary units are of vague or indeterminate value, the statistical results may perhaps be plausible, but they certainly will be untrustworthy. In other words, a statistical table is a sort of numerical syllogism, in which everything depends upon the premises. It is for failing to recognize or observe this fact that some statisticians have misled, rather than instructed, the scientific reader on the subject of the diseases of occupations. Especially is this so as to the effects of certain occupations in increasing certain diseases. Let us take the hypothetical case of the cotton-spinners, and suppose that a general claim was made that their occupation predisposed to pulmonary tuberculosis. Let us suppose that tables are prepared showing the rate of death for cotton-spinners the world over from tuberculosis, and that from these a general average was struck. The result in such a table would be of little if any value in determining the question of the influence of their occupation upon the genesis of tuberculosis. The reason is that the primary factors or units are of unknown or indeterminate value. These primary units are simply so many deaths of individual cotton-spinners from tuberculosis. The important questions of heredity, of locality, of race, of country, of hygiene, of habits, and of infection are all left untouched. These, as predisposing or exciting causes of tuberculosis, would require in turn at least as careful a statistical treatment, for purposes of supplying control-tables, as did the mere coincidence of the victim's occupation and disease.

If now we take Hirt's table on the relative totalities of phthisis among various classes of workers in dusty occupations ("Krankheiten der Arbeiter," p. 30) we obtain more trustworthy and rather striking results. This table compares the figures for the various classes, *i.e.*, those working in metallic dust, in mineral dust, in vegetable dust, etc., with one another. The result shows a much higher percentage for those working in metallic dust. The primary factors here have an approximately known value (because it is determined without statistics that working in dusty occupations produces phthisis), and the statistical conclusions are not for the purpose of proving that already well-known fact but to present side by side the figures for the prevalence of this disease in the various divisions of dusty occupations, and so to find in which division this disease is most prevalent. The table does this, and nothing more. It allows a fair inference to be drawn that working in metallic dust is the most conducive to phthisis of these various occupations, but it does not absolutely prove this.

The same may be said of Givre's statistics for tuberculosis among the workers in silk in Lyons ("De la Tuberculose chez les Ouvriers en Soie," 1890). This work was suggested by the prevailing belief in Lyons that silk-workers are more exposed to tuberculosis than are other artisans. Givre's statistics show that the percentage of deaths from tuberculosis in silk-workers to deaths from all causes is not much in excess of this percentage in the general population (25.1 per cent. for the former to 20.2 per cent. for the latter) and that it is not so high as for some other occupations, notably that of tailors. The primary factors, or units, in this table, however, are not of absolutely known value, hence the table is not absolutely demonstrative. For instance, for aught we know, the industry of silk-weaving may draw for special reasons from a section of the population which from constitution or heredity is specially predisposed to tubercle; or, on the other hand, the contrary may be the case, and persons of exceptionally good constitution may alone be willing to engage in the work. In the former case, the percentage might indicate that silk-weaving is rather beneficial to consumptives; in the latter, that it is frightfully mortal to healthy men.

One of Farr's tables in the Registrar-General's reports ("Supplement," p. xxxv., 1864) shows the average mortality per cent. of males aged fifteen years and upward in various occupations in the years 1860–61 at different periods of life. Various trades and occupations are grouped in this table, as tailors, shoemakers, farmers, butchers, workers in iron, copper, tin, and lead, and others. The table compares the percentages of mortality for these various groups in decades from fifteen to eighty-five years and upward, with the general mortality for all males in England for similar periods. It takes no account of *causes* of death, nor of the various conditions of living, as locality, prosperity, prevalence of epidemic disease, heredity, race, etc. And yet in this table the primary factors or units are, if not of known, yet of approximately equal value, because it may be supposed that in such general divisions of the population the influences of locality, heredity, race, disease, etc., will average about the same. Hence the results of this table are not without interest and value. The table shows, for instance, an exceedingly high rate of mortality for inn and hotel keepers, publicans, and beer-sellers, as compared with that for most other occupations, notably for grocers and butchers. It is noteworthy that in the decade from forty-five to fifty-five years their mortality percentage is greater than for men working in iron, copper, tin, or lead.

The elaborate study of the mortality of males engaged in different occupations, by Dr. William Ogle (Supplement to the Forty-fifth Annual Report of the Registrar-General of England, 1885) is one of the best examples of the statistical method and its legitimate results. Dr. Ogle states fairly the difficulties that beset this method. Among these are the vagueness of the statement of occupation (and we may suppose of disease) on the mortality returns. One of the most serious, however, is due to the following ever-acting cause: Many occupations require a certain high standard of health to permit men to follow them successfully, those, for instance, of a blacksmith or miner; just so soon as health and strength fall below the standard necessary to permit a man to compete in such occupations, he must retire; he then usually takes up some lighter toil, or retires altogether; the weaker individuals are thus being constantly weeded out, and go to swell the lists of common or general laborers, street-sellers, or men without work. Hence the mortality rates for the occupations requiring heavier toil are unfairly lowered, and the rates for general laborers, etc., are increased to an appalling degree. In this criticism Dr. Ogle, it seems to me, has put his finger upon one of the weakest spots in the statistical method, and one that can be detected almost everywhere. Again, as he says, the various trades do not start on equal terms; a weak man will not become a blacksmith, a miner, or a navvy, but will take a lighter trade, as that of a tailor or weaver. Hence the heavier trade seems to derive an advantage. It may be said in reference to this, however, that this advantage may be more than compensated for by the fact that the heavier ttrade wears men out more rapidly; in other words, a strong man, a a railroad navvy, may not have so good a chance for prolonged life as a weak man may as a tailor. In all these instances we see that the primary factors of the statistics are of very uncertain value.

When, however, Ogle shows by statistics that inn-keepers die from an unusually large percentage of diseases of the liver (seven times as high as for males in general) his result is striking, and it must be convincing that alcohol plays an important part in the mortality of this class.

In the present paper the statistical method will not be used, unless by reference to the labors of others in that field. Where reliable results seem to have been obtained by experts and by those having access to large bodies of figures, these results may be incorporated in the text, with due ackowledgments. Instead of the statistical method this paper will follow the descriptive method. This latter has the advantage of dealing more directly with facts, and of presenting them in a form more convenient and serviceable for the practising physician. Thus the effects of lead-poisoning, and the symptoms of "grinders' asthma," are presented in the concrete, without reference to generalizations based upon statistics or mortality returns. These latter more concern the sanitarian, who must contemplate disease in a more generalized form and must combat it in the gross.

AGE AND SEX IN OCCUPATIONS.

In estimating the general effects of occupations on health it is essential to note carefully the important factors of age and sex. Statisticians as a rule are careful to do this. In a descriptive paper, such as this, in which the main reliance is not based upon statistics, it is equally important. These factors vary within wide limits, not only according to the country but also according to locality and even according to the kind of occupation. This variation is so great, in fact, that it is not possible here to present anything like a complete study of the subject. Reference is made to it in this place simply to call attention to its importance, so that observers of these diseases, who consult this article, may not be unmindful of these factors, and may have a few facts by way of illustration.

Arkwright's invention of the "spinning-frame," following on Hargrave's invention of the "spinning-jenny," caused a complete revolution in the cotton industries, which up to that time had been pursued with the simplest apparatus from time immemorial. The distaff and the spindle, which were in use in every cottage all over the civilized world, as they had been from the dawn of history, were replaced by ingenious machinery which vastly surpassed their simple capacity and greatly increased the demand for labor. Two immediate and lasting effects of this change were the building of factories and the use of child-labor. So great was the demand for this labor in the north of England that the apprentice system with its worst evils was greatly extended. When the cupidity or necessity of parents in the rural districts could no longer meet the demand, children were transported from the large cities and distant towns. The workhouses were the sources of much of this labor. It is said that in one instance an agreement was made between a London parish and a Lancashire manufacturer stipulating that with every twenty sound children one idiot should be taken. These children were sadly abused and overworked. A system, in fact, soon sprang up which equalled, if it did not surpass, some of the worst features of negro slavery. Children of nine and ten to thirteen and fourteen years of age were forced to toil at monotonous and confining labor for as much as sixteen hours a day. The hour, nominally given to dinner, was taken by these poor wretches to clean and oil their machines. The factories in which they worked were, before the introduction of steam, usually placed in the country beside a stream for the purpose of obtaining power, but after the introduction of steam these mills began to be aggregated in closely built towns. Hence the hygienic conditions became generally bad. The factories themselves were ill built and ill ventilated; they were overheated and the air was sometimes laden with dust. The moral guidance which these apprentices received was practically naught; their food and clothing were cheap and poor.

It was against such a system and its environments of uncouth and comfortless-looking tenements that the English poet Southey inveighed, and sighed in vain for a return to a more primitive England when every woman sat and spun in her own vine-covered cottage; but it was in its favor that Macaulay, the essayist, criticising Southey's book,* attempted to prove the superiority of the factory-system by the fact that the poor rates were less in the manufacturing than in the rural districts, apparently ignoring the fact that the factories had taken away a part of the farmer's means of support. It was this system that led one observer to look upon the introduction of extensive manufacturing establishments as an evil, "accumulating the fruits of labor within the possession of a comparatively small number of capitalists, to the utter destruction of all health and comfort on the part of the actual producers of wealth."†

The natural effects of such a system were soon apparent. A factory population was growing up in England whose future and whose future progeny gave statesmen and moralists alike grave concern. When at last the devastation of epidemic disease added to the existing horrors of the system, legislators could ignore the subject no longer. In Manchester, according to Professor Robertson, the fearful spread of epidemic disease made havoc among the youthful laboring population because of their scanty living and methods of work. These were mostly pauper children from the south of England who were housed and fed in the most shocking way. Their wretched state finally made some impression even upon the minds of the extreme school of political economists who believe in individualism in industrial affairs. The parliament of Great Britain was appealed to at last with success, and the principle was established of the "right of the state to control the industrial organizations which depend upon the labor of women and children." The first "factory act" was passed in 1802. The second was Peel's act in 1819. Since that time the legislation in Great Britain has been full and far-reaching, and has apparently led that of all the civilized world, to which it has given strength and example. Those interested in the history of the legislative control of female and child labor in manufacturing establishments will find the subject presented with full details in Prof. Robertson's article on the "Factory Acts" in the "Encyclopædia Britannica."

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* See Macaulay's Essays.

† Consult Dr. J. O. Green's paper on the factory system, 1846.

By the provisions of the act of 1819 children were not to be employed before the age of nine, and between that age and sixteen were restricted to twelve hours of labor a day, exclusive of an hour and a half for meal times. By another act night work was prohibited in the cotton mills for persons under twenty-one years of age. By the act of 1833 night work was prohibited to children under eighteen in cotton, wool, worsted, hemp, flax, tow, and linen spinneries and weaving-mills; children from nine to thirteen were not allowed to work more than forty-eight hours a week; and youths from thirteen to eighteen were restricted to sixty-eight hours a week (Robertson). Provision was made for school attendance. Mining industries likewise were afterward restricted in the employment of child-labor. These acts were bitterly opposed for economical reasons.

This factory legislation in England culminated in an act in 1878. A presentation of the full scope and details of this legislation cannot be given here. Some of its more important features, however, are as follows:

In textile factories, *for young persons and women* the hours are from 6 A.M. to 6 P.M., or from 7 A.M. to 7 P.M.; on Saturday from 6 A.M. to 1 P.M. for manufacturing purposes, and to 1:30 for all employment, if one hour is allowed for meals; otherwise work shall be stopped at 12:30 and 1. If, on Saturday, the work begins at 7 it shall stop at 1:30 and 2 P.M. respectively. For meals two hours on week-days and one-half hour on Saturday are allowed. Continuous employment without a meal shall be for a period not to exceed four hours and a half.

For children, employment is to be for half-time only, in morning or afternoon sets, or alternate days. A child must not be employed for two successive periods of seven days in the same set, whether morning or afternoon, nor on two successive Saturdays, nor on Saturday in any week if on any day he has been employed for more than five hours and a half. A child shall not be employed on two successive days nor on the same day in two successive weeks (Robertson).

These regulations vary somewhat for non-textile factories, but their spirit and scope are practically the same. The reasons for, or the wording of, some of the regulations are not very clear, as, for instance, why a child should not work on the same day in two successive weeks. Inspectors and medical examiners are appointed to see to the proper enforcement of these laws.

Women also have been brought under the protection of some of these regulations; the attempts of political economists, even in England, not having been successful in combating them. Of their wisdom and efficacy I cannot speak.

Of the necessity for, or desirability of, laws regulating child-labor evidence in the affirmative is furnished by Dr. J. S. Torrop in a recent paper on "Factory Children" (quoted by Arlidge). This observer was certifying surgeon, *i.e.*, the official appointed according to English law to examine the physical state of laboring children, and he has given some significant facts in his interesting paper. Of 2,000 children examined by him 1,771 were specimens of what he describes as the ordinary "factory child." The average weight of these Lancashire factory children was not equal to the average elsewhere. The low average is much more marked among children of thirteen, who have already served three years in the mills, than in those of ten years of age. Of 60 of these children the average weight was eighteen pounds below the average of good health. Among the poorest of this group were many defective and diseased children, numbering so high as 198, or about ten per cent. Dr. Torrop says, "The promising child of ten entering the factory degenerates into the lean and sallow person of thirteen, and this process continues until a whole population becomes stunted, and thus the conditions of life in factory towns become a real source of danger to England's future. In addition to the loss of physique it is instructive to note the deterioration in personal appearance. Out of the 2,000 children under notice, only sixteen could be described as handsome, and of these the larger portion were girls from Ireland."

It must be borne in mind that in the cases of children it is not only severe toil and long hours that do harm. Monotonous and uninteresting labor, with deprivation of amusement and healthful exercise, defects in or total want of education, and poor food and clothing, are direct causes of the startling degeneration of this class. This evil, which has now been growing for little more than a century in the older countries, must present to future generations many grave results and anxious problems.

The conditions of labor vary so much in different countries that deductions must not be hastily drawn from data supplied by one and then applied to the other. In the United States, as is well known, up to the present time, labor of all kinds has enjoyed extraordinary advantages. This is due partly to the fact that our population is still hardly equal to all the demands made upon it, hence to the high price of labor; and partly to the factitious value placed upon labor by our protective tariff. Child-labor, as known in England and some continental countries, is scarcely known here. The conditions formerly existing, especially in some of the New England towns, were almost ideal. Dr. John O. Green, in 1846, wrote a paper in which he presented a truly roseate view of mill-work and mill-life in Lowell, Mass., claiming that there was no strictly factory population in that busy city. Statistics by the Rev. H. A. Miles (before 1846) showed that female operatives in Lowell did not work in the factories on an average for more than four and one-half years. He claimed that a model moral hygiene prevailed there; intemperance and immorality not being tolerated. This population was recruited largely from rural New England. These young women were healthy to begin with; they did not commence work at an early age when they were not fitted to endure it; they were moral, educated, and, in a sense, independent. They adopted this remunerative labor in order to secure a modest competence, and having secured it were satisfied to quit. The chief causes of ill-health among them were to be found in improperly cooked, not deficient, food. This satisfactory state existed in New England a half century ago, and the traditions of it probably still linger there. It is not to be expected, however, with the changes in industrial conditions and policy, and with the increasing population and the cheapening of labor, that such an ideal factory population has been, or at least can continue to be, maintained.

Lévy, basing his statement on observations in France, says that women suffer more from the effects of occupation than do men, and that the mortality among working women is higher than that among men of the same class. This statement is contrary to recent conclusions reached in the English Registrar-General's reports, which are to the effect that the mortality of male workers exceeds the mortality of female workers at nearly all ages. The truth is that all such generalized statements are apt to be misleading. Men suffer a higher mortality because they enter upon more laborious and more dangerous occupations than do women, hence statistical conclusions are of no great value. Woman may suffer in health in numerous ways that do not affect materially her mortality. For instance, neurasthenia, the bane of overworked and underfed women, does not leave a definite trace on the mortality tables. Again, woman's ill health and drudgery in a factory may affect her progeny in a way that the statistician cannot estimate. My own observation, not so much among mill-hands as among female clerks, shop-girls, school-teachers, and music-teachers, is that these suffer more readily from a mild grade of neurasthenia than do young men, as a result of an equal degree of work and strain.

Women and children suffer in special ways from some occupations. Both are rather more liable than men to pulmonary affections caused by working in a dust-laden air. Women are said to suffer from uterine derangements and displacements from occupations requiring long standing and certain movements of the body, while the lacteal secretion is impaired by some occupations, and also by enforced absence of mothers from their nursing infants. In this connection a recent writer, H. H. Champion (*The Nineteenth Century*, October, 1890) says, significantly, that mill-owners prefer married women to girls as mill hands "because mothers with children to feed would put up with almost anything rather than lose their work!"

The relations of the factory system to the system of education, and their conjoint action upon the health of factory children, constitute an important topic which is admirably discussed by Mr. T. P. Sykes (*Fortnightly Review*, December, 1889), in a paper entitled "The Factory Half-Timer." According to this writer the factory child of tender years, who rises at five o'clock in the morning and after a long walk works until the afternoon, is not in a bodily or mental condition to receive into his sleepy brain the dry elements of a routine education which is forced upon him in the latter part of the day. The elementary school under such circumstances is "a crushing, cramming, grinding-according-to-the-code establishment."

In France the laws regulating child-labor are complete. A recent writer in the *Bulletin de la Société de Protection des Apprentis et des Enfants Employés dans les Manufactures* (Jan., Fév., Mars, 1894, 137) says that the time is happily past in which it was necessary to combat for the amelioration of the physical conditions of apprentices and of the work of children in factories. The recent laws of 1874 and 1892 insure this. According to these laws "the age of admission into factories has been raised, the duration of daily work has been reduced, work at night has been abolished as much as possible, the physical aptitude has been declared obligatory, the regulations relative to hygiene and to the security of workmen have become more strict, and, finally, a powerful organized body of inspectors secures the strict observance of the law."

The special society, whose journal is quoted above, was founded by G. B. Dumas in 1866. It has been decreed of public utility (July 4th, 1868). Its object is to ameliorate the condition of apprentices by all means which, while respecting the liberty of the employer and the authority of the parent, act in conformity with the law. It is thoroughly organized and equipped, and has distributed much money in its work. The journal of the society, which is specially devoted to this cause, is a valuable periodical.

LEGAL RESTRAINTS UPON THE EMPLOYMENT OF WOMEN AND CHILDREN IN THE UNITED STATES.

*A Synopsis of the Laws of the States of Pennsylvania, New York, Massachusetts, Maryland, Connecticut, Delaware, New Jersey, and Ohio.**

No restraints, such as indicated by the title, are known to common law. As far as existing, they are all founded on statute.

As the subject is not within the jurisdiction of the Federal Government, no general statutes of the United States treat of the subject.

A considerable number of the different States, and more especially of the manufacturing States, have, however, enacted special statutes upon the subject in question.

These statutes are not exactly identical in any two of the States, but they are all founded on one of the following principles:

A. Employment of women should be restricted, where such employment would tend to deprave the morals of others.

B. Restraints should be put upon the employment of children and—to a certain extent—of women, where such employment would tend to injure the employés themselves by endangering either:

1. Their moral purity, or
2. Their life or physical health.

These principles are not directly expressed in any of the said statutes, but one or more of them will be found underlying them all.

In some cases, it will not be possible to separate the rules according to these principles, as they are founded on more than one of them. They will be treated of under one or the other heading, according to what principle seems to have been the principal moving cause for their enactment.

A.—*Restraints upon the Employment of Women, where such Employment would Tend to Deprave Others Morally.*

Pennsylvania.—Under the *Act of May 22, 1879*, § 2, it is unlawful for any female to attend among, or wait upon the audience or spectators at any exhibitions, performances, or entertainments, such as mentioned in § 1 (s.: all kinds of theatricals, varieties, minstrelsy, ballets, circus, and the like), or at any other place of amusement in any city of the first class (cities containing a population of 600,000 or over; Philadelphia is the only one of that class within the commonwealth) to procure, offer, furnish, or distribute any description of commodities or refreshments whatsoever. All persons are enjoined from the employment of any female for any such purpose.

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* This digest was made at my request specially for this paper by Axel Teisen, Esq., of the Philadelphia bar.

Violations are punishable by fine, not exceeding $500, or by imprisonment not exceeding one year, or both, or either.

Pennsylvania.—Under the *Act of March 28, 1878*, § 1, no female may be employed as waiter girl in inns or other places where intoxicating drinks are sold.

§ 2 fixes the punishment, for each girl so employed, at fines, not less than $100 and not exceeding $500, or imprisonment not less than three months nor exceeding twelve months, or either, or both.

§ 3 directs that in addition the violator shall have his license forfeited.

Maryland.—Public General Laws, Act 27.

§ 85 makes it unlawful to employ or allow female sitters in any variety establishment or concert hall.

§ 86 makes violations punishable by fine, not less than $200 nor exceeding $1,000, or by imprisonment in house of correction from two to twelve months, or both, for each female sitter.

§ 253 makes it unlawful, in the city and county of Baltimore, to employ female waiters in any theatre, museum, or other place of amusement.

§ 254 fixes the punishment for violations at fines not less than $100 nor exceeding $1,000, or by imprisonment in county jail from one to six months, or both.

B.—*Restraints upon the Employment of Children and Women for the Benefit of such Employés.*

1. *To Hinder their Moral Depravement.*

a. Children.

Pennsylvania.—The *Act of June 11, 1879*, § 2, makes it a misdemeanor to procure employment for, or employ any *minor under* the age of *fifteen years*, for rope or wire walking, or as an acrobat, gymnast, contortionist or rider, or for any obscene, indecent, or illegal exhibition or vocation, or any vocation injurious to the health or dangerous to the life and limb of such child engaged therein; or to retain, harbor, or employ *any minor* child in or about any assignation house or brothel, or in any place where any obscene, indecent, or illegal exhibition takes place. Violations are made punishable by fine, not less than $50 nor exceeding $100, for each offense.

§ 4 makes it a misdemeanor to procure employment for or employ any *minor under* the age of *fiften years*, to sing, dance, act or in any manner exhibit, in any dance house whatever, or in any concert saloon, theatre, or place of entertainment where wines or spirituous or malt liquors can be obtained. Violations punishable by same fine.

§ 3 makes it as a misdemeanor to procure employment for, or to employ any *minor under* the age of *eighteen years* for the purpose of singing, playing on musical instruments, begging, or for any mendicant purpose whatever, on the streets or other highways of the Commonwealth. Violations punishable as above.

The *Act of April 21, 1849*, makes it a misdemeanor to employ *children between thirteen and sixteen* years of age in any cotton, woollen, silk, paper, bagging, or flax factory for more than nine calendar months within any one year, unless they shall have attended school for at least three consecutive months within the same year. Violations punishable by fine of $50.

New York.—By the laws of 1886, chapter 409, § 4, as amended by *Laws 1889, chapter 560*, § 4, it is made unlawful to employ any *child under* the age of *sixteen years*, who cannot write or read simple sentences of the English language, in any manufacturing establishment, except during the vacations of the public schools of the city or town of residence of such child.

Penal Code, § 292, as amended by *Laws 1884, ch. 46 and Laws 1886, ch. 31*, makes it a misdemeanor to procure employment for, or employ *any child actually or apparently under* the age of *sixteen years*, for the purpose of exhibition:

1. As a wire and rope walker, dancer, gymnast, wrestler, contortionist, rider or acrobat, or upon a bicycle or any other mechanical vehicle or contrivance.

2. In begging or soliciting or receiving alms in any manner or under any pretense, or in any mendicant occupation, or in gathering or picking rags, or collecting cigar stumps or bones or refuse from markets.

3. In peddling, singing, or playing upon a musical instrument, or in a theatrical exhibition, or in any wandering occupation.

4. In any illegal, indecent, or immoral exhibition or practice, or in the exhibition of any such child when insane, idiotic, or when presenting the appearance of any deformity or unnatural physical formation or development.

5. In any practice or exhibition or place dangerous or injurious to the life, limb, health, or morals of the child.

Penal Code, § 289, as amended by *Laws 1888, ch. 145*, § 5, makes it a misdemeanor to wilfully cause or permit *any child actually or* *apparently under* the age of *sixteen years* to be placed in such a position, or engage in such an occupation, that its life or limb is likely to be endangered, or its health to be injured, or its morals to become depraved.

The Laws of 1887, ch. 552, § 1, make it a misdemeanor for persons and corporations employing *messenger boys* to place instruments of communication, or permit them to remain, in any disorderly house or any unlicensed saloon, inn, tavern, or other unlicensed place where wines or liquors are sold.

§ 2 makes it unlawful for such persons and corporations to send any *messenger boy* to any such place. §§ 3 and 4 make violations punishable by fines of $50 for each offense.

Massachusetts.—Public Statutes, 1881, ch. 48, § 8, makes it unlawful to employ or exhibit, or to suffer to be employed or exhibited, any *child under* the age of *fifteen years* in dancing, playing on musical instruments, singing, rope or wire walking, riding, or performing as a gymnast, contortionist or acrobat, in any circus or theatrical exhibition or in any public place whatever. Violations are made punishable by fine, not exceeding $200, or by imprisonment in county jail for not more than six months.

Public Statutes, 1888, ch. 348, § 1, make it unlawful to employ any *child under* the age of *thirteen years* in any *indoor work,* performed for wages or other compensation, during the hours when the public schools are in session, and to employ such child in *any manner,* unless it shall have attended school for at least twenty weeks in the year next preceding such employment.

§ 2 makes it unlawful to employ any *child under* the age of *fourteen years* in any factory, workshop, or mercantile establishment, except during the vacations of the public schools, unless it shall present a certificate of such school attendance as required by § 1.

Maryland.—Public General Laws, Art. 27, § 273, make it unlawful to employ *children under* the age of *sixteen years* for singing, playing on musical instruments, rope walking, dancing, peddling, begging, or any mendicant or wandering business whatsoever. Violations made punishable by fine, not less than $50, nor exceeding $250, or by imprisonment in the county jail from thirty days to one year, or both.

Connecticut.—General Statutes, 1888, § 2105 (Laws 1882, ch. 80 and 1885, ch. 90), make it unlawful to employ any *child under* the age of *fourteen years,* having resided nine months within the United States, at any labor, unless it shall have attended a day-school during at least twelve weeks or sixty full school days of the twelve months preceding any month in which such child shall be so employed, and at least six weeks thereof must have been continuous. Violations are punishable by fine not exceeding $60. § 2106 makes it the duty of the employer to procure from the proper teacher a certificate of school attendance. Under § 2107 parents or others, violating the provisions of § 2105 by making false statements concerning age or term of residence in United States, or by instructing child to make such false statement, are punishable by a fine not exceeding $7, or by imprisonment for not more than thirty days.

Delaware.—Revised Code, 1852, as amended by *Laws 1887, ch. 150, vol. 16* (March 18, 1879, § 2), make it unlawful to procure employment for or employ any *child under* the age of *fifteen years*, for rope or wire walking, or dancing, or as an acrobat or gymnast, or for begging, or any obscene, indecent, or illegal exhibition or vocation, or any vocation injurious to the health or dangerous to the life or limb of such child engaged therein, or to retain, harbor, or employ *any minor child* in or about any assignation house or brothel. Violations are punishable by fine, not less than $20, nor exceeding $100.

New Jersey.—Revision, 1887, Labor, § 10 (P. L., 1883, p. 59, March 5, 1883) makes it unlawful to employ any *child between* the ages of *twelve and fifteen years* in any factory, workshop, mine, or establishment where the manufacturing of goods is carried on, unless, during the twelve months next preceding such employment, it shall have attended some school for five days every week, during at least twelve consecutive weeks.

Ohio.—Revised Statutes, 1892, § 4394, make it unlawful for any pawnbroker to employ any *minor under* the age of *sixteen years* to take pledges in pawn.

§ 6986 makes it unlawful to employ any *child under* the age of *fourteen years* in singing, dancing, playing on musical instruments, rope or wire walking, riding, or performing as a gymnast, contortionist, or acrobat, in any circus or theatrical exhibition. Violations punishable by fine, not exceeding $200, or by imprisonment for not more than six months, or by both.

B.—1. *b. Women.*

Pennsylvania.—The *Act of June 3, 1893,* § 1, makes it the duty of persons and corporations employing women, to provide separate, proper, clean, and well-ventilated wash- and dressing-rooms and water-closets for females, entirely away from those used by the men.

New York.—Laws 1886, ch. 409, § 13, added by Laws 1887, ch. 462, § 4, and amended by Laws 1889, ch. 560, § 7, make it the duty of persons and corporations employing women, to provide separate and suitable washrooms for females; to make the water-closets, intended for the use of women, separate and apart from those used by men, and properly screened; also to provide dressing-rooms for the female employees, when required by factory inspectors to do so. Violations are punishable by fine, not less than $20, nor exceeding $100, or by imprisonment from thirty to ninety days, or by both.

B.—2. *For the Protection of the Health of Female and Minor Employés.*

a. Seats for Female Employés.

Pennsylvania.—The *Act of March 22, 1887*, § 1, makes it the duty of all persons and corporations employing females in any manufacturing, mechanical, or mercantile establishment to provide suitable seats for the use of the female employés, and to allow them the use thereof when not necessarily engaged in the active duties for which they are employed. Under § 2 violations are punishable by fine, not less than $25, nor exceeding $50, or by imprisonment in case fine is not paid.

Massachusetts.—Laws 1882, ch. 150 (April 12, 1882, § 1).

Verbatim as above § 1.

§ 2 imposes a fine for violations of not less than $10, nor exceeding $30.

Delaware.—Revised Code, 1852, as amended 1887, ch. 238, vol. 18, § 1 (April 21, 1887).

Verbatim as above § 1.

§ 2 imposes, for violations, a fine of not less than $25, nor exceeding $50.

New Jersey.—Revision 1887. Health, § 147 (P. L., 1882, p. 227) makes it the duty of persons employing females in any mercantile business or occupation to provide and maintain suitable seats for them, at or beside the counter or work bench, and to permit the use of such seats to such an extent as may be reasonable, for the preservation of their health.

§ 148 imposes, for violations, a fine not exceeding $100.

B.—2. *b. Factories and Mines.*

Pennsylvania.—The *Act of June 3, 1893*, § 1, makes it unlawful to employ *any minor* or to detain him at labor in any manufacturing establishment or mercantile industry, or in any laundry or renovating establishment, for more than *twelve hours* in any *one day*, or for more than *sixty hours* in any *one week.*

§ 2 prohibits the employment, in any of the said establishments, of any *child under* the age of *thirteen years.* Employers of *minors under* the age of *sixteen years* must keep a record of their name, birthplace, age, place of residence, name of parent or guardian, and the time when the employment ceases. These facts to be stated in an affidavit by parent or guardian (or by child itself, if there be no parent or guardian), to be made and filed with the employer, before any employment must take place.

§ 3 makes it the duty of all employers of *women and children, or either*, in any manufacturing or mercantile establishment, or renovating works or laundry, to keep posted conspicuously in every room where such help is employed, a printed notice, stating the number of hours per day, for each day of the week, required of such persons, and in every room where *children under* the age of *sixteen years* are employed, in addition a list of their names with their ages.

§ 4 defines the words "factory, manufacturing and mercantile establishment" and states that places where less than five persons are employed shall not be deemed such within the meaning of the act.

§ 5 makes it the duty of the Governor to appoint, every three years, a *factory inspector*, whose duty it shall be to visit and inspect all factories, work-shops, and other establishments employing women and children; also to prosecute all violations of the act; also to report to the Governor once every year.

§ 8 prohibits the employment of any *minor under* the age of *sixteen years* in the cleaning of machinery while in motion.

§ 17 makes violations of act punishable by fine, not exceeding $500.

The *Act of May 24, 1893*, prohibits the employment of any *minor under* the age of *fourteen years* to have the care or management of an elevator. Violations are made punishable by fine, not less than $25, nor exceeding $100.

The *Act of June 11, 1879*, § 5 makes it a misdemeanor to employ any *child under* the age of *twelve years* in any mine or underground work or like place whatever.

Violations are made punishable by fine, not less than $10, nor exceeding $50.

Under the *Act of June 2, 1891*, § 1, no *boy under* the age of *fourteen years*, and no *female of any age* must be employed, or permitted for the purpose of employment, in any mine. No *boy under* the age of *twelve years*, and no *female of any age*, must be permitted in the outside structures or workings of a colliery, for the purpose of employment, except in office work.

§ 2 makes it the duty of the employer, in case of doubt as to age, to procure proof thereof by certificate from parent or guardian.

The *Act of May 15, 1893*, prohibits the employment of *boys under* the age of *twelve years* and of *females of any age* in bituminous coal mines; no *boy under* the age of *sixteen years* must mine or load coal, unless in company with a person over sixteen years of age.

New York.—Laws 1886, ch. 409, § 1, as amended by Laws 1889, ch. 560, § 1, and Laws 1892, ch. 673, § 1. *Males under* the age of *eighteen years*, and *females under* the age of *twenty-one years* must not be employed at labor in any manufacturing establishment for more than *sixty hours* in any one *week*, nor for more than *ten hours* in any one *day*, except for the sole purpose of shortening hours on Saturdays; the said employés must not be employed at labor in the hours between nine o'clock P.M. and six o'clock A.M.

§ 2 as amended by Laws 1887, ch. 462, § 1, and Laws 1889, ch. 560, § 2. *Children under fourteen years* of age must not be employed at labor in any manufacturing establishment. Persons employing *children under sixteen years* of age must keep a register, showing the name, birthplace, age, and place of residence of all employés under the age of sixteen years. No person shall be permitted to employ children under the age of sixteen years, unless he shall have first procured and placed on file an affidavit by the parent or guardian of such child (or by the child itself in case it has no parent or guardian) stating its age and place of birth. Said register and affidavits to be open to the inspection of the factory inspector, his assistants and deputies.

§ 3, as amended by Laws 1892, ch. 673, § 3. In manufacturing establishments, employing *boys under eighteen years* of age and *females under twenty-one years* of age, there must be posted, in every room where such help is employed, a notice showing the number of hours per day, for each day in the week, required, and where *children under sixteen years* of age are employed, in addition a list of their names with their ages. No *child under fifteen years* of age must have the care, custody, management of, or operate any elevator, and no *person under eighteen years* of age must have the care, custody, management of, or operate any elevator running more than 200 feet a minute.

§ 4, as amended by Laws 1889, ch. 560, § 4. The factory inspector shall have the power to demand, for *children seemingly feeble*, a certificate of physical fitness from some regular physician; if such is not obtainable, then he shall have the power to prohibit the further employment of any such child.

§ 6. The Governor shall appoint a *factory inspector* and one assistant factory inspector, whose duty it shall be to visit and inspect all factories, to report to the Governor once a year, to enforce the provisions of the act, and to prosecute violations thereof.

§ 11, added by Laws 1887, ch. 462, § 4, prohibits the employment of any *female under twenty-one years* of age and of any *male under* the age of *eighteen years* in the cleaning of machinery while in motion.

§ 20, added by Laws 1887, ch. 462, and Laws 1889, ch. 560, § 8, makes every violation of the act a misdemeanor, punishable by fine, not less than $20, nor exceeding $100, or by imprisonment in the county jail from thirty to ninety days, or by both.

§ 21, added by Laws 1887, ch. 462. A copy of the act must be kept posted in each workroom of every manufacturing establishment or mercantile house where there are employed persons affected by the act.

Laws 1892, ch. 673, § 7 defines a manufacturing establishment as "any mill, factory, or workshop where one or more persons are employed at labor."

Massachusetts.—Laws 1888, ch. 348, § 1. *Children under thirteen years* of age must not be employed in any "factory, workshop, or mercantile establishment."

§ 2. No *child under fourteen years* of age must be employed in any manner during the hours between seven o'clock P.M. and six o'clock A.M. The chief of police, with the approval of the Governor, may designate any kind of work as injurious to the health of children under fourteen years of age, and after one week's written notice given to the employer, no child under fourteen years of age shall be employed thereat.

§ 3. No *child under sixteen years* of age must be employed in any factory, workshop, or mercantile establishment, unless the employer first shall have procured and placed on file a certificate, stating the name, age, residence, and place of birth of such child, and also keeps a full list of such children so employed.

§ 9 fixes the punishment for violations at fines, not less than $20, nor exceeding $50, or at imprisonment in the county jail for not more than thirty days, or both.

Laws 1882, ch. 74, § 4, as amended by Laws 1887, ch. 280, § 1 (*May 17, 1887*). No *minor under eighteen years* of age and no woman must be employed in laboring, in any manufacturing or mechanical establishment, for more than ten hours in any one day, except as hereinafter stated, and in no case shall the hours of labor exceed sixty in a week.

There shall be posted in a conspicuous place, in each room where such persons are employed, a printed notice, stating the number of hours' work required of them on each day of the week, the hours of commencing and stopping such work, and the hours when the time or times allowed for dinner or other meals begins and ends.

Exemptions from ten-hours rule: (1) for the sole purpose of making a shorter day's work for one day in the week; (2) to make up for time lost on some previous day of the same week, in consequence of the stopping of machinery, upon which such person was employed or dependent for employment. Such stopping must have lasted at least thirty minutes, and a written report thereof must have been previously sent to the chief of police or factory inspector, before any such overtime employment shall thereby be authorized.

§ 5. Violations of § 4, including the making of false reports concerning the stopping of machinery, are made punishable by fine, not less than $50, nor exceeding $100.

Laws 1887, ch. 215 (April 21, 1887). All *children, young persons and women*, employed in the same factory, shall be allowed their meal time or meal times at the same time; provided, that if any of them begins work at a later hour than the rest, he may have his meal times at other times, but must not, during regular meal hours, be required to tend the machinery, or do the work of other children, young persons, and women, in addition to his own.

§ 2. *Children, young persons, and women* must not be employed for more than six hours, without an interval of one-half hour for meals. Still they may be employed for six and one-half hours, if the work ends at one o'clock P.M., and they are then dismissed for the day—or for seven and one-half hours, if the work ends at two o'clock P.M., and they are then dismissed for the day, and an opportunity is given them to eat a meal in the mean time.

§ 3. Exempt from the rules of §§ 1 and 2 are: Iron works, glass works, paper mills, letter-press printing establishments, print works, bleaching works, and dyeing works. The chief of police shall have power to exempt such other works, where labor must be kept up continuously, when such exemption will not injure the health of the children, young persons, and women employed.

§ 5. Violations are made punishable by fine, not less than $50, nor exceeding $100.

Laws 1888, ch. 426 (May 29, 1888), § 1. *Women and children* shall not be employed in any factory, workshop, or mercantile establishment in a room above the second story, from which there is only one way of egress, in case the inspector of factories shall so direct.

Maryland.—Public General Laws, Art. 27, § 139. No *child under sixteen years* of age must be employed in laboring in any cotton, woollen, or other manufacturing establishment for more than ten hours in any one day.

§ 140. Violations are made punishable by fine, not exceeding $50 in each case.

§ 141. The foregoing provisions do not apply to agricultural or household work, nor to mercantile establishments.

Connecticut.—General Statutes 1888, § 1745 (1887, ch. 62). No *minor under sixteen years* of age and *no woman* shall be employed in any manufacturing, mechanical, or mercantile establishment for more than ten hours in any one day, except when machinery must be repaired to prevent interruption of continuous working, or for the sole purpose of making one shorter day of work in the week. Notice of the number of hours required every day of the week must be posted in a conspicuous place in every room where such persons are employed. In no case must such persons be employed for more than sixty hours during any one week. Violations are punishable by fine, not exceeding $20.

§ 1753 (Laws 1886, ch. 124). *Children under thirteen years* of age shall not be employed in any manufacturing, mechanical, or mercantile establishment.

§ 1754 (ibid.). Violations are punishable by fine not exceeding $60. Every week of such employment shall be considered a distinct offense.

§ 2263–2272. The Governor shall, every two years, appoint a factory inspector, whose duty it shall be to inspect the factories, to report to the Governor once a year, to enforce the factory-acts, and to prosecute all violations of the factory-acts.

New Jersey.—Revision 1887. Labor. (P. L. 1883, p. 59, March 5, 1883.)

§ 9. No *boy under twelve years* of age and no girl *under fourteen years* of age shall be employed in any factory, workshop, mill, or establishment where the manufacturing of goods is carried on.

§ 11. No *child under fourteen years* of age shall be employed in any factory, workshop, mill, or establishment where the manufacturing of any goods is carried on, for more than an average of ten hours a day, or sixty hours a week.

§ 12. It shall be the duty of the *inspector of factories*, to be appointed by the Governor, to enforce the provisions of the above sections.

§ 17. The inspector of factories and his deputies shall have the power, in the case of a *child seemingly unfit* to do the work he is employed to perform, to demand a certificate of physical fitness from some regular physician, and in case such certificate is not obtainable, they shall have the power to prohibit the further employment of such child.

§ 26. Violations of the foregoing sections are made punishable by a fine of $50 for each offense.

Ohio.—Revised Statutes 1892, § 6986. No *minor under twelve years* of age shall be employed in any factory, workshop, or establishment wherein the manufacturing of any goods of any kind is carried on.

§ 6986 *aa.* No *minor under eighteen years* of age shall be employed in any of the places named, for more than ten hours during any one day, and in no case for more than sixty hours in a week. Notice of the hours required every day of the week must be posted in a conspicuous place in every room where such persons are employed.

§ 6986 *bb*. Violations are made punishable by fine, not less than $50, nor exceeding $100, or by imprisonment in the county jail from thirty to ninety days.

§ 6986 *c*. It shall be the duty of the *inspector of factories and shops* to enforce the foregoing provisions.

Relations of Race and Country to the Diseases of Occupations.

This subject offers many problems of interest, few if any of which can be solved here. It is, in fact, doubtful whether they can be solved accurately by any writer with the materials at hand. Such a solution requires an intimate knowledge of the racial characteristics and the habits and customs of various countries which is possessed by few if any. It is probable in fact that customs, laws, habits of the people, food, sanitary arrangements, and the available markets have more to do with this question of occupation-diseases than has the question of mere race. Arlidge quotes, apparently with approval, an opinion of Greenhow that the Welsh and the Celtic Irish suffer in a higher ratio from their occupations than do the English, but the statement is open to doubt. The food supply is of prime importance in any estimate of the mortality of a laboring population, but that is foreign to our subject which is strictly concerned with the direct effects of occupation. In Germany, for instance, it is said that a weaver in former days was able to afford meat, and that the cheapest, only on one day in the week. In the far East the bulk of the population live on rice; on which food it may be doubted whether they can ever compete, as some economists have feared they yet will do, with the laboring masses of Europe and America.

Classification.

As already said, only those effects of occupation will be described in this paper that are special and distinct. Those artisans only will be considered who by their occupations are exposed to a definite risk, to which, as a rule, others are not exposed. Hence the subjects to be classified are limited in a number of ways and in a variety of directions. A purely sanitary study, for instance, based upon the various hygienic elements that enter into the health account of every workman, will not be made. Neither will every disease that may afflict artisans be considered: this, for obvious reasons, takes from the subject all individuality. Finally, accidents from machinery, explosives, etc., will not be considered: they constitute a purely surgical subject, and, so far as I know, have nothing especially distinctive in them.

Even, however, with the subject thus limited, it is not easy, nor possible, to devise a classification that is perfectly satisfactory, or at least free from faults. Three methods are possible: First, by the occupations themselves. This appears, at first sight, the most natural; and it is the one that has been adopted by most writers, from Ramazzini down. It is open, however, to many objections. It lends itself too readily to a discursive treatment of the subject—a fault that is apparent in the writings of most of those who have used it. They give a catalogue of trades and avocations followed by a description of many inconsequential diseases. It thus leads to conclusions that are often too broad and sweeping. But its greatest fault, in a condensed paper, is the fact that it renders necessary much repetition. Thus, in the case of lead-poisoning, it is necessary to describe again and again the symptoms of this toxæmia under each heading of lead-miners, lead-makers, file-cutters, painters, etc. A second scheme is to classify according to the diseases. A moment's reflection, however, shows that this plan is not sufficiently distinctive: a workman, for instance, may suffer from a variety of diseases, one of which may be due to his trade, another only partly due to it, and another quite independent of it. Besides, for reference, such a plan is the least valuable, because it leads to classifying members of the same group of workmen under different headings; as for instance, the disorders of painters would classify some of these artisans under the head of gout and others under the head of chronic lead-poisoning; and finally because it presupposes a knowledge of the subject before such a plan can be handled or consulted with ease. The third plan is to classify according to the causes of disease; and this is at once the most natural and most convenient. This is so because it is logical and rational to approach the study of a subject by studying first its causes; and because, secondly, while this plan does not avoid entirely the necessity for some repetition, it is not so objectionable on that score as the other methods. If, for instance, we take one of the metallic poisons we can group under this heading all the trades involved, with a brief account of each; if one trade is involved with more than one cause (as brass-founders are said to be exposed to both copper and arsenic) this fact can be pointed out by a cross-reference. By this plan, the symptomatology, which, with cure and prevention, is the most important part of the subject, does not need to be described more than once. Finally, this plan reduces to a secondary place the consideration of technical trade-details, and advances to first importance the noxious agent itself. This is an important consideration, because this paper is written from the medical or pathological standpoint, and cannot enter fully into minute details of the various manufacturing processes, to describe which requires knowledge that is not at the command of a physician. The medical practitioner is interested to know the cause of the disease and what are its effects; the exact manufacturing process that set it agoing being of secondary importance to him. Enough of these trade-details will be described, however, to render the subject intelligible.

This etiological plan admits of a division under three headings.

(1) *Injurious Substances.*—This group includes by far the largest number of occupation-diseases that will engage our attention. Injurious or poisonous substances, by being introduced into the body, cause many of the most distinct and easily recognizable of these affections. It is customary to subdivide them according to their nature (as whether they are metallic, mineral, animal, etc.) or again as to the manner in which they gain admission into the body (as by inhalation, ingestion, absorption, etc.). After mature reflection I have decided not to do this, for several excellent reasons. For instance, as to the first-named plan, it matters little whether such substances as arsenic, mercury, etc., be called "metals" or "minerals;" the distinction is rather arbitrary. Some injurious substances, as certain kinds of dust, are in fact compounds of many mineral and metallic matters; while others are compounds of animal, vegetable, and earthy substances. For statistical purposes, such subdivisions may be necessary, but hardly for a simple descriptive paper. As to the second-named plan, it need only be said that most poisonous substances are introduced into the body in a variety of ways; as, for instance, lead, which can be inhaled as fine dust, or swallowed as paint, or absorbed directly through the skin. The substances in this group will be taken in a simple order as they stand naturally related to each other, and briefly described; the trades engaged with them will then be briefly mentioned and the manufacturing process for each indicated; then the symptomatology, which is mainly the same for each trade using identical poisons or exposed to the same injurious substances, will be described in full; and, finally, cure and prevention will be discussed.

(2) *Injurious Environment.*—A large number of the diseases of artisans are caused by defective or unwholesome surroundings. Such general insanitary conditions as bad ventilation, over-heated air, and insufficient light are common causes of these diseases. The difficulty is to estimate them at their true value. In many instances this

cannot be done and no attempt to do it will be made here. In some instances, however, the cause is so apparent that it can be taken fully into account, as in the over-heated air of some mills and factories and of mines. Here again, however, the subject dovetails into that of dust and other injurious substances; but this is inevitable. Then there are special instances that furnish most interesting phenomena of disease, as the compressed air of caissons, and the violently concussed air of boilers in process of being rivetted. These and allied subjects will be taken up seriatim and treated in the same manner, so nearly as can be, as that indicated for the first class.

(3) *Injurious Exercise.*—A certain small, but important, section of the diseases of occupations is composed of those that are caused by over-exercise of some particular part or parts. Scrivener's palsy is the most familiar of these. Others are the cramps and palsies of telegraphers, piano-players, engravers, etc., and the aphonia of clergymen and other voice-users. In this class may be comprised those who suffer from constrained positions while at their work. In each instance the special action of the cause will be considered first; after which the symptoms, pathology, and treatment will be described in due order.

With reference to the sources of information for this work, I desire to say that the technical details have been obtained from the best and most recent authorities, as well as from some personal observation. As, however, the processes of manufacturing are changing constantly, and vary even in different countries and localities, I cannot vouch always for the accuracy of the description of them. This knowledge lies somewhat outside of the field of a physician's observations. When possible I have made direct observations in factories and mills. The pathological details are gathered almost exclusively from the writer's own observation. In an experience of years in several large hospitals and clinics in Philadelphia I have seen and studied a large number of the diseases of occupations.

1. Effects of Injurious Substances.

EFFECTS OF ARSENIC.

Metallic arsenic occurs native in the rocks in many regions, as in the silver mines in Chili. The principal ore is the arsenical pyrites. Arsenic is found also in tin, copper, cobalt, and nickel ores. It is sublimed from the ores by roasting, the arsenic going over and condensing in appropriate chambers or flues. The salts of this metal are much used in the arts and in medicine. It is said that in one year more than 1,800 tons of white arsenic (arsenious acid) came from one mine in Derbyshire, England. Arsenic acid is made from arsenious acid by treating the latter with strong nitric acid. It is less poisonous than arsenious acid; in fact, persons employed in its manufacture are said to increase in weight. In manufacturing arsenic the chief danger to the workmen is in opening the flues in which the arsenic has condensed. According to some, however, the smelters seldom suffer in well-kept establishments (Tracey). Both the fumes and the dust are sources of danger if precautions are neglected. In some processes of smelting, as in Cornwall, where the tin and copper ore is rich in arsenic, the workmen are said to be more inconvenienced by the sulphurous than by the arsenious fumes. The millers and grinders of the salt, coming into direct contact with it, get ulcers and eczema (Bowhay).

Arsenic acid was formerly, and may be yet in some places, used to produce some of the brilliant aniline dyes, especially magenta. Aniline is an artificial alkaloid, itself not without poisonous properties; but some of its supposed ill effects are said to be caused by arsenic and other acids with which it is combined. Recently, by amended processes, the use of arsenic acid as an oxidizing agent is said to have been dispensed with (Arlidge). The symptoms of aniline-poisoning proper will be noted under the appropriate heading.

The most important preparations of arsenic in the industrial world are the two copper salts, known as Scheele's green, and Schweinfurt or Vienna green. The former is an arsenite of copper; the latter an aceto-arsenite of copper. They are both beautiful green pigments. Because of their tendency to blacken in foul gases and when mixed with oil they are more useful to the paper-stainer than to the painter (Paton). They have been used extensively in staining wall-paper, in coloring toys, and in coloring artificial flowers. Grinding Schweinfurt green is considered a dangerous operation; although, according to Hime, the workman suffer but little, in spite of being covered with the dust from head to foot.

In some processes for printing wall-paper the design is first printed in strong size; metallic powders are then sprinkled by hand over the paper. When Schweinfurt green is used thus the workman is of course exposed to the dust arising from the process, and may be poisoned. Paper-hangers may even be affected by the arsenic in some wall-papers, especially when scraping off old papers. Thirty years ago numerous instances occurred of persons being poisoned from occupying rooms thus papered (Beaugrand). It is but fair to say that several manufacturers of wall-papers testified some years ago before a commission of the Massachusetts legislature that during many years' experience they had never known of harm from arsenic to themselves or their employés. Such evidence, however, must be taken with allowance. Other papers, especially fancy papers for wrapping and for decorative purposes, are colored with these pigments. In some instances the colors are put on in a moist state, by which process they are less likely to affect the artisan. After drying, however, small particles and dust are given off, and may infect the air. Cutter has reported instances of arsenical poisoning in employés in a box factory where arsenical papers were used.

The manufacture of artificial flowers is a most dangerous trade, from its liability to cause arsenical poisoning. The business has much increased in recent years, especially in England, France, and America. In England, according to one authority, 4,886 people were engaged in it in the year 1871. Much popular notice was called to it, several years since, because of the evil arising from the arsenic used in it. The majority of artisans employed in this industry are women and girls. They sometimes work at home, but more frequently in small shops; the custom varying, probably, according to country and town. In France it is said that between 2,000 and 3,000 shops exist for the manufacture of artificial flowers. The stage of the process which causes danger is coloring the leaves and petals. After these have been cut out by a stamp, then moulded into shape by hand, then mounted on wires which form the stalk or stem, they are colored. When Scheele's green is used it is dusted on the flower, which has previously been gummed or waxed over. This dusting is done from a dredging box held in the hand, and of course gives rise to much loose dust, which floats about in the air. According to Tracey the hands of artificial flower-makers are stained yellow with green finger-nail tips. The yellow is caused by picric acid; the green by the arsenical salt, which lodges under the nails. It is said that in recent years the use of arsenical pigments is not so common in this industry.

These green arsenical colors are used also in lithographic printing. They are sometimes used dry, being dusted on a prepared surface. In this process, and in the subsequent work on the presses, the workmen are exposed to some danger.

Arsenic is mixed with lead for making shot; when thus made the shot falls round or "truer" than when made of lead alone. In the latter case the shot is likely to be elongated or "tailed."

Among objects contaminated with arsenic may be mentioned stockings, veils, cretonnes, and other prints, red-striped bed-ticking, Turkey red cotton, some woollen dress-goods, hangings, and curtains, and anything in which arsenical aniline dye is used. These dyes contain two per cent. of arsenic by weight (Shattuck). Arsenite of soda is used as a mordant to fix color. Putnam observed an epidemic of arsenical poisoning in an infant asylum in which the poison was found in some dresses recently supplied. Shattuck relates an interesting case of arsenical poisoning in a physician from playing-cards, the lesions being confined largely to the hands. Arsenic is used also by taxidermists.

The *symptoms* of arsenical poisoning are either acute or chronic. Acute arsenical poisoning is rare among artisans who work with the poison. These acute symptoms, as is well known, resemble somewhat those of Asiatic cholera. There is the same sudden seizure with vomiting, purging, pain in the abdomen, cold extremities, rapid pulse, and collapse. Nervous symptoms, as paralysis and coma, sometimes supervene before death. For a description of this acute poisoning the article on toxicology in this volume may be consulted. We are more concerned here with the chronic poisoning, which is the form usually seen in workmen.

It is well known that chronic symptoms may follow, after an interval, acute symptoms. Shattuck suggests that this may be due to the storing up in the system of the albuminoid compounds of arsenic, which are then gradually set free. E. S. Wood found arsenic in the urine of a man for ninety-three days after the acute stage had subsided. Arsenic is stored more in the brain and cord than in the liver.

Chronic poisoning by arsenic affects especially the respiratory and gastro-intestinal tracts, the skin, and the nervous system.

Dryness and irritation of the mucous membrane of the nose, eyes, mouth, throat, larynx, and bronchi are seen. Nausea, loss of appetite, and other evidences of obstinate dyspepsia appear. Diarrhœa, with colicky pains, is experienced. The discharges from the bowels become mucous in character—a significant sign. With these gastro-intestinal symptoms great languor and emaciation supervene, and the patient may even become hectic. A mild grade of this chronic poisoning is sometimes borne for a long while, as seems to be the case with some makers of artificial flowers.

Associated sometimes with this gastro-intestinal derangement is an irritation of the bronchial mucous membrane; this is so especially when dust and fumes of arsenic prevail. In the Cornwall works, however, this is attributed by Bowhay to the fumes of sulphur (sulphurous acid) which issues in large quantities from the furnaces, the tin ore being rich in sulphur as well as arsenic.

The skin suffers severely in some instances of chronic arsenical poisoning. Thus, according to Bowhay, the grinders of white arsenic have eczematous sores and ulcers of the skin. The eczema is commonly present in the folds of the neck and around the nostrils. A scratch or abrasion grows rapidly into an ulcer (Arlidge). According to Kirchgässer a deposit of pigment in the skin of the face is a symptom of chronic arsenicism. Lepine also described a pigmentation of the skin, sometimes called "arsenical bronzing." Beaugrand, in 1859, claimed that the arsenical green pigments, then much used in coloring wall-papers and artificial flowers, caused a characteristic eruption on the skin of the artisans. This skin affection resembled some of the eruptions of syphilis. The dermatosis is vesicular, then pustular, and is seen especially about the nose, mouth, and chin, and about the genitalia, these various parts being infected by the hands.

Rasch (*Annales de Dermatologie et de Syphiligraphie*, 1893, IV., 150) has described the various dermatoses of arsenical origin, including in his paper purpura, pigmentation, erythema, and vesicular and urticarious eruptions. Hardaway and Fox (*British Journal of Dermatology*, 1893, V., 304), each have described a keratosis of the palms and soles due to arsenical poisoning.

Irritation of the kidneys is observed in chronic arsenicism. It is shown by albumin, casts, and even blood in the urine.

Some of the most characteristic symptoms of chronic arsenical poisoning are observed in the nervous system. Arsenic leads gradually to a diffuse or multiple neuritis. This neuritis causes weakness of the extremities, progressive atrophy of the muscles, abolished tendon reflexes, and the reactions of degeneration to the faradic and galvanic currents. The sensory nerves also are involved, with resulting anæsthesia especially in the extremities of the limbs. Pain is experienced on pressure over the nerve-trunks, and the muscles are sore on pressure and handling. Trophic sores, noted by some, are probably due to the local action of the poison on the skin. Other important nervous symptoms are tremor and ataxia. The tremor is caused by arsenic, just as it is by lead, mercury, and alcohol, by interference with the nutrition of the nerve-centres. The ataxia is that of a pseudo-tabes, *i.e.*, due to the involvement of the sensory nerves, instead of a posterior sclerosis. Arsenic may cause also headache, vertigo, tinnitus aurium, depression of spirits, convulsions, neurasthenia, and hysteria. Some of these more obscure nervous symptoms are in fact most apt to perplex the diagnostician, and must be scrutinized and weighed with great care. Toxic hysteria is one of these; so important, indeed, is it that a special discussion of it will be in order after the description of the various poisons. The anæsthesia caused by arsenic is sometimes profound; in one case, known to the writer, the patient, who was semi-delirious, also owing to the poison, declared his belief that his legs were cut off because he could not feel them.

Arsenic, in chronic poisoning, can usually be found in the urine.

According to some recent experimenters, whose results, however, are denied by others, arsenic checks tissue-change.

As stated already, it is claimed that the workers in arsenic acid grow fat. This belief partakes somewhat of the nature of a legend. It is customary to refer to the peasants of Styria as examples of arsenic-eaters who thrive on the poison. According to Maclagan, who visited Styria (*Edinburgh Medical Journal*, 1864), the custom really exists; but, according to a royal commission which investigated the subject, it has been greatly exaggerated. Arsenic-eating, in very small doses, is not strictly analogous, however, to the absorption of the fumes or dust by artisans. The toxicophagi probably stimulate the mucous membrane of the stomach, and so promote appetite and digestion, with a regulated dose—conditions that do not obtain in slow poisoning by inhalation.

The *treatment* for these various effects of arsenic must be twofold: first, by directing the patient to abstain from the labor that occasions them; second, by relieving individual symptoms.

The first indication is absolute in most cases, and is the only treatment needed. A workman who is suffering from chronic arsenical poisoning must quit his work, unless some efficient means of protection can be devised. If he is willing to quit, his health will usually recover itself without much medical treatment.

The symptomatic treatment for chronic arsenical poisoning is not necessarily so varied as the symptoms. The gastro-intestinal and the cutaneous lesions usually yield promptly when the cause is removed. The weakness and emaciation tend to subside likewise; at most they require only good food and good hygiene. The nervous symptoms are not so readily controlled. The paralysis, myalgia, neuralgia, and anæsthesia require time for their cure. Strychnine hypodermatically is a valuable remedy, and massage, electricity, the phosphates, and cod-liver oil are important adjuvants. There is no antidote nor specific for chronic arsenicism.

More important than cure is prevention. Workmen in arsenical substances of any kind ought to be protected from the dust and fumes as much as possible. In addition, those whose hands are covered with dust ought to exercise scrupulous cleanliness before eating; the hands should be thoroughly washed and the finger-nails cleaned. Some of the poison no doubt gains entrance into the mouth and stomach by the neglect of this rule. Frequent bathing and change of underclothing should be observed. When practicable the hands might be protected with gloves—but of the practicability of this there is considerable doubt. It would always be better to use Scheele's and Schweinfurt greens in a moist state if this could be done, instead of by dry dusting, as has been the practice in some arts in the past.

According to some observers there seems to be a relative immunity among workmen. But is it real? Workmen are afraid to complain or to give testimony against their employers for fear of losing work. There is no reason for believing that the system becomes inured to the poison by constant exposure to it—at least in mills and factories, where the dose cannot, from the nature of things, be regulated.

There are laws regulating or prohibiting the use of arsenic in some arts in Sweden, Germany, Russia, Austria, and Denmark. Legislation on the subject is needed in all countries. It is possible that agitation on the subject in past years, and the fear of damages, have restricted the use of arsenic in the arts to a considerable extent.

Effects of Mercury.

Mercury, for all practical purposes, is obtained from cinnabar, which is a sulphide of the metal. Cinnabar is found in many countries, but, until recent years, the largest supply came from Almaden in Spain. Profitable mines exist also at Idria in Illyria. The Spanish mines, which have been worked from antiquity, were for centuries the source of most of the mercury of commerce. In recent years, however, they have been eclipsed by the quicksilver mines of California. Thus the United States, mainly California, produced 2,054 tons of mercury in 1875 to 929 tons produced by Spain in 1873.

Metallic mercury may be recovered from cinnabar either by simple roasting or by reducing the ore with iron or lime, a sulphide of the latter elements being formed. In the first-named method the mercury is volatilized; and, in the crude process still used at Almaden, much of it is lost, and goes to destroy vegetation and to injure the health of the workmen. So repulsive is the work that the Spanish Government, according to Tardieu, has had great difficulty sometimes in obtaining workmen to engage in it. Convicts have been employed, but not with good results. In some instances, by granting them unusual privileges, such as exemption from taxation and conscription, men were induced to engage in the work. The miners suffer as well as the smelters. The air of the mines contains mercurial vapors, as is proved by an amalgam forming on bright gold objects carried in the pockets of visitors. Some of the evils at Almaden, it seems, might be prevented by better hygiene. I am not able to say what is the state of health of the workmen in the California mines. Information of that subject is much desired.

Mercury, it must be recalled, is readily volatilized, and to this fact is due much of the damage that results from it in all industries in which it is used. In fact every artisan who comes in contact with it in any way in the course of his work incurs a risk from it on account of this quality. This volatility begins at a temperature of −13° C. (8.5° F.) and increases with heat.*

Mercury is used extensively for extracting the precious metals, gold and silver, from their beds or ores. The ores are worked up with mercury so that the metals form an amalgam; this amalgam is then separated from the dross. It is then subjected to distillation, by which the mercury is volatilized and the gold or silver is left. The technical processes vary in different places and countries. In the case of gold, lumps of the solid amalgam are distilled in vessels lined with fire-brick and wood-ashes, heat being applied with care that the retort does not become red-hot. The mercury, thus driven off, may be recovered and used again. In the case of silver, the process for making the amalgam in Mexico has some unique features. The ore is crushed and mixed with certain reagents, and mercury is added, the mass being trodden under the feet of mules until perfect amalgamation is effected (Ditmar). This process requires from fifteen to forty-five days. Its effects upon the health of the workmen and of the mules must be deleterious in the extreme.

In the processes for gilding and silvering these amalgams with mercury are used. The old method of fire-gilding is still considered the best, although it is extremely dangerous to the artisan. An amalgam of gold with mercury is spread upon the surface to be gilded; the mercury is then driven off by heat, the gold being left as a close, firm, and durable layer. For silvering, the process is practically the same. An amalgam is made with mercury and applied with a brush to the baser metal, which is to be silvered. The mercury is then volatilized as in gilding. The silvered surface is very durable and need not be thick to stand high polishing. This method is expensive and dangerous and is said to be almost superseded by electroplating. In these old processes for gilding and silvering the dangers to the workmen were great. In fact few if any escaped mercurial poisoning; the artisans found it necessary to leave the work for seasons or to abandon it altogether. For bronzing, an amalgam of mercury, tin, and bismuth is used, with similar evil results.

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* The incident, mentioned by Taylor in his work on poisons, may be recalled of a British vessel, carrying as part of her cargo a large quantity of mercury, becoming permeated with the fumes from the metal, which had escaped from its receptacles in the hold. Many persons on board, as well as animals, were seriously poisoned.

The makers of felt hats suffer severely sometimes from the effects of mercury. This has been noted for many years, in Germany especially. Attention was called to it in this country in 1859 by Dr. J. A. Freeman, of Orange, N. J. ("Mercurial Disease among Hatters," Trans. N. J. Med. Soc., 1859, p. 61), who had noted the prevalence of mercurial poisoning among the hatters of Orange, Newark, Bloomfield, and Milburn, in that State. Later (1878) Dr. L. Dennis ("Hatting as Affecting the Health of Operatives," Report of the Board of Health of New Jersey, 1878) made an elaborate report on the subject, based especially on a study among the extensive hat factories in Newark, N. J. The cause of the mercurial poisoning in hatters is readily understood and can be explained without going too far into the technique of the trade. Felt is made of the hair or fur of various animals, especially the beaver, rabbit, hare, and coney; lower grades are made with various admixtures of wool and even cotton. It has been found by experience that these furs blend and adhere better in felting after having been treated with a strong solution of acid nitrate of mercury. Dr. Dennis, from microscopic studies, was led to believe that the mercurial salt increased the natural roughness of the serrated surface of the hair, thus inducing a more thorough and intimate felting. This process of treating with nitrate of mercury is effected by brushing; it does not appear to be dangerous to the workmen. The danger arises at a later stage, when the felt is being made into hats, or during the finishing. At this time the felt, impregnated with the mercurial salt, is subjected to heat, and is worked often in a warm, close room. Hence mercurial vapors rise and affect the operatives. Dr. Freeman noted that the poisoning was always worse in cold weather, *i.e.*, when the men worked in warm rooms with closed windows. In some of the earlier stages much dust is given off in mixing and sorting the fur. This dust is an irritant to the respiratory organs, and being charged with mercurial particles is all the more injurious. It is noteworthy that, according to the table of death-rates compiled by Dr. Ogle in England, for the years 1880–81–82, the mortality rate for hatters was rather high, being 1,064 to 1,000 for all males. In discussing this fact, however, Ogle does not refer to the possible effect of mercurial poisoning.

The makers of thermometers, barometers, and other philosophical instruments containing mercury, are occasionally poisoned with it. These artisans are few in number, and, according to Whitely, do not suffer as a rule. In their cases there may be absorption through the skin as well as inhalation of the vapors.

In former times mirrors were made almost entirely with an amalgam of tin and mercury. In recent years, however, the finest mirrors are made by depositing on the glass a coating of pure silver. The old amalgamation-process, which first made the Venetian mirrors so famous and the evil effects of which on the health of the workmen are referred to by Ramazzini, consisted in spreading out a piece of tinfoil, somewhat larger than the glass, then "quickening" this with mercury (really forming an amalgam), then pressing the plate of glass down upon the sheet of amalgam and holding it there under weights. Subsequently the surface was exposed and dried (Paton). This process, as can readily be appreciated, was disastrous in the extreme to the health of the artisans from the mercurial vapors liberated.

Vermilion is much used as a pigment. It is a sulphide of mercury, and as such is found native, as already explained. Very little of the vermilion used in the arts, however, is the native ore (cinnabar). It is an artificial product obtained by mixing sulphur and mercury. The first product of this mixture is the *black* sulphide of mercury, from which vermilion is made by a process of distillation in pots, the brilliant scarlet sulphide adhering to the walls of the vessels. It is subsequently ground. This sulphide of mercury is not readily absorbed, and probably is not responsible for much if any poisoning. In the process of distillation from the black sulphide it is possible that mercurial fumes are given off; but of any evil effects upon the workmen I have no knowledge. Vermilion is used by dentists for coloring the plate for holding false teeth. When introduced for this purpose the question arose whether the metal could be absorbed in poisonous quantities. Prof. S. H. Guilford, of Philadelphia, tells me that no authentic instance is on record of such poisoning. He gave me this opinion with reference to the case of a woman who claimed that several sores on her gums, caused by an ill-fitting set of teeth, had been induced by mercurial poisoning from the vulcanite plate, and who threatened to sue her dentist.

Mercurial salts, especially the corrosive chloride, are much used now in medicine, surgery, and obstetrics for antiseptic purposes. Physicians, surgeons, and nurses, who use them often and in strong solutions, are exposed to some slight risks. Instances of poisoning, however, are extremely rare.* The local effect upon the hands is sometimes that of an irritant.

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* A. W. Foot (quoted by Wood, "Therapeutics," 8th ed., p. 530) reported a case of paralysis of the muscles of the hand and forearm from absorption of the red iodide of mercury during the act of rubbing cattle with a salve containing this salt.

Mercurial salts, like arsenic, are used for preserving the skins of animals for stuffing. They are also used by furriers.

The *symptoms* of chronic mercurial poisoning, the form usually presented by artisans, are stomatitis, salivation, ulceration of the gums, necrosis of the bones of the jaw, falling of the teeth, fetor of the breath, emaciation, anæmia, digestive disorders, affections of the kidneys, a characteristic tremor, and paralysis. Moreover, the victims of hydrargyrism are likely to transmit to their offspring cachectic defects, as rickets, etc. Acute symptoms are not common among artisans; vomiting, diarrhœa, and inflammation of the kidneys, which follow the ingestion of poisonous mercurial salts, are the chief among these acute symptoms that are rather conspicuous by their absence.

The affection of the mouth presents an acute and chronic stage, as Roussel noted among the workmen in the Spanish mines. The acute form is identical with that which is seen sometimes after the medicinal use of a mercurial. The mucous membrane of the mouth and throat is inflamed, the gums are swollen and ulcerated, the salivary glands are congested, salivation is intense, and the enlarged tongue is scarcely held within the limits of the teeth. This acute form was seen by Roussel in novices at the mines and furnaces. The chronic form follows upon repeated acute attacks, or, more frequently, is due to a slow, gradual action of the poison. This was the more common form seen by Roussel at Almaden, and is the form usually seen in all workmen. These accidents are sometimes primary, without a precedent acute stage. The gums, perhaps formerly ulcerated, heal and recede; the teeth are uncovered and become loosened, and are lost one by one; the odor of the breath is insupportable. In the most chronic cases the victim has no pain or fever, no swelling of the salivary glands; there is perhaps for a while only slight ptyalism. In some the gums become swollen and bleed easily. The termination in all cases is the same, *i.e.*, the loss of the teeth. According to Roussel, the miners think this a happy issue from their troubles; they believe that when a miner loses all his teeth, or even only his molars, he is thenceforth exempt from suffering so far as his mouth is concerned. Many of these Spanish miners are toothless by the time they are thirty-five years of age. In the most severe forms of ptyalism, of the acute variety, exfoliation of the jaw-bones is sometimes seen, but this does not appear to be a common accident in the chronic mercurial poisoning of workmen.

Emaciation and anæmia are usual accompaniments of a severe grade of chronic hydrargyrism. Roussel speaks of young men of twenty-three at Almaden with the figures of old men. Artisans, exposed to mercury, are usually pale, the women abort easily, the girls are chlorotic, and the children "scrofulous." The albumin, fibrin, and red blood corpuscles are diminished in mercurial poisoning. A condition known as "mercurial scurvy," characterized by anæmia, wasting, prostration, diarrhœa, fœtid breath, falling of the hair, rheumatoid pains in the bones and joints, and œdema, is sometimes seen.

The kidneys are violently inflamed in severe acute poisoning with mercury. In the chronic forms lesions of the kidneys do not appear to be so common as one would expect from the action of the poison in the acute variety. In a case of acute poisoning with corrosive sublimate in a dog I once saw the kidneys much swollen and inflamed.

Some of the most characteristic effects of mercury are those that it causes in the nervous system. The most common are tremor, paralysis, and mental changes.

Mercurial tremor usually begins insidiously in those who are exposed to the vapors of the poison; it is not a symptom or aftereffect of acute poisoning. In artisans it may have been preceded by some of the changes already noted in the mouth; but occasionally it is primary in those whose health otherwise is fairly well preserved. It is both favored originally and intensified by alcohol and tobacco. It begins as a rather fine tremor, most marked at first in the upper extremities; later it spreads to the voluntary muscles of other parts, as the face, neck, tongue, and legs. As it spreads it tends to increase in amplitude, *i.e.*, it becomes jerky and somewhat choreiform. It is increased by emotion and by volition; but it is not always absent during repose. Hence it is not exactly like either the tremor of paralysis agitans or that of disseminated sclerosis. The speech is often affected. In severe cases the tremor interferes seriously with voluntary motion; the patient cannot feed himself and can scarcely walk. It ceases, as a rule, during sleep. According to Tardieu the curious fact has been noted in Spain that the tremor ceases in the interior of the mines. This tremor, in some cases, has a distinctly hysterical character and setting, as will be discussed in a future section.

The paralysis caused by mercury is, as a rule, of the type of a peripheral neuritis. In some cases it is quite local, *i.e.*, confined to one or a few nerves; in others it is much more diffused. In chronic hydrargyrism, as Vicente de Arevaca pointed out long ago, intense pains in the extremities are common. According to his description (quoted by Tardieu) these pains appear to be distinctive of a neuritis. They occur usually after the tremor is established; and, if the work is not abandoned, they become almost intolerable at times. They begin sometimes in the great toe or in the thumb, and spread from one or the other of these points. They are intense and lancinating. They are also the cause of the insomnia that plagues these victims. As they continue, the loss of power becomes apparent. Among local palsies noted are those of the extensors of the wrist (as in lead-poisoning) and of the muscles of the larynx.

Sensory changes other than pain are seen as various types of anæsthesia, some of them distinctly hysterical, as shown by the fact that they may be influenced and even abolished by suggestion (Letulle).

Mercury produces a somewhat characteristic encephalopathy. The old observers at Almaden noted especially stupidity and loss of memory. According to Kussmaul inability to fix the attention, irritability, and insomnia are seen. In some cases hallucinations and delirium, constituting a true confusional insanity, are present.

Finally the effects of chronic mercurial intoxication in the offspring must not be ignored. There can be no doubt of this hereditary influence. Kussmaul ("Untersuchungen über den constitutionellen Mercurialismus") has discussed this subject at length. Children born of women thus poisoned are often feeble, ailing, rickety, and seem to be prone to tuberculosis. Goetz saw an infant with congenital tremor (Beaugrand). Women who work at making mirrors frequently abort. Among noteworthy facts are the following: Children born when their parents were not working in mercury were healthy; children born of the same parents who had become workers in mercury were diseased. Lizé has noted this heredity among the children of hatters.

As in the cases of arsenic and all poisons used in the arts, the *treatment* is not so important as the prevention of the poisoning. Unfortunately, it seems that in some industries in which mercury is used this prevention cannot be absolute. On the other hand much evil is due to the carelessness and bad habits of the workmen. As the timid Hindu will walk unconcernedly in his garden which is infested with the deadly cobra, so will many artisans go into or stay recklessly in the fumes of mercury. Christison refers to two barometer-makers who slept all night in a room in which a pot of mercury was giving off its fumes. Dennis found an appalling indulgence in alcohol among the hatters of Newark, N. J. The subject of prevention, moreover, involves such a knowledge of trade technique that very frequently the physician can do nothing more than point out the manifest evils and recommend all possible care; while the details must be left to builders, mechanics, and proprietors, who are familiar with the operations involved. It may be laid down as a general rule that any scheme for abating the nuisance arising from mercurial vapor must depend upon ventilation. Plans must be devised for carrying off the fumes. Freeman, as said above, found that the evil effects among hatters were worse in cold weather, when these artisans worked with closed windows.

The precautions that are time-honored in the quicksilver mines might be observed with profit by all artisans who use mercury. After quitting work a bath should be taken and the clothing changed. The workmen should not eat, nor even drink, while at work. It is best that they should sweat as little as possible while at work, because a moist skin favors absorption; and that they should rest as little as possible in the fumes, because repose after active labor is supposed to facilitate absorption. Of course they should never sleep in a room in which there are the slightest possibilities of the presence of mercurial vapor. A temporary change of work, when possible, from one involving exposure to the fumes to another in which there is no exposure, is desirable. Thus, at Amalden, the miners are sometimes sent to do agricultural labor.

The medicinal treatment for chronic mercurial poisoning is not promising. The patients tend to recover from some of the symptoms if they quit work; if they do not quit no medicine can avail them. Some of the more chronic symptoms, however, such as tremor, may persist in spite of abandoning work and of the use of any or all medication. Chlorate of potash is recommended usually for the affections of the mouth; its results, however, are not always brilliant. In a case recently under my care of severe salivation with necrosis of the upper jaw, due to an hypodermatic injection of calomel, nothing but time and frequent washings with hydrogen peroxide seemed to do good. The elimination of mercury is said to be increased by the iodide of potassium. The remedy should be given in small doses (3 to 5 grains) gradually increased. Sulphur baths are said to increase the amount of mercury excreted by the kidneys (Gowers). Sulphur may be given also by the mouth. It is considered by some to be of first importance.

EFFECTS OF LEAD.

Lead, of all the industrial poisons, is the most important, because of the diversity of its uses and the wide spread of its evil effects.

Lead exists in several forms in Nature. The most common is the ore known as galena, which is a sulphide of the metal. Galena is widely diffused, and is the principal lead-ore of Europe. In this country it is found especially in Missouri and the upper Mississippi valley. In Nevada and Colorado immense deposits of carbonate of lead are found. This ore is rich in silver (one to two per cent. at Leadville), for the sake of which it was originally worked. Of late, however, the production from it of lead has been of commercial importance. Galena is so insoluble that it is not so common a cause of poisoning in the miners as might be supposed. That it may be injurious, however, is proved by the fact that fish cannot live in water polluted with the washings (Jackson). Hirt states, moreover, that almost all the miners in Saxony show the blue line on the gums. Poisoning among Spanish miners also appears to be observed (Bages, *Gaceta Médica*, Madrid, 1851, VII.; also *Journal de Chimie Médicale*, 1852). Jackson says (*British Medical Journal*, July 25th, 1857, p. 619) of the lead miners of Yorkshire that they inhale dust laden with lead. He claims that, as a rule, they do not have paralysis and colic, because lead in galena is not very absorbable. Yet their average duration of life, according to him, is only from forty-five to fifty years.

Webb (*British Medical Journal*, Aug. 15th, 1857) says that the lead-miners of Derbyshire are known by their pallid faces, and that they have such symptoms of plumbism as anæmia, prostration, constipation, headache, rheumatoid pains, and the blue line. And yet he says, rather inconsistently, that they are a healthy, long-lived race. Wrist-drop is not common among them.

From galena metallic lead is obtained by roasting, the suphur being burned away as sulphurous acid and the lead remaining. The processes for lead-smelting vary; but all of them involve the production of a "lead-smoke" (oxide and sulphate of lead, finely divided), which is caused by the volatility of lead at a red heat. This smoke may be, and often is, the cause of poisoning. Smelters, according to Jackson, have an average duration of life eight to ten years less than miners.

After smelting, lead is still contaminated with iron, antimony, zinc, silver, and arsenic, and must be refined. Refining is done with varying processes, chiefly oxidizing under heat, and may be the source of plumbism.

Rolling sheet-lead is a source of danger. In rolling and folding the sheets cutaneous absorption takes place. This happens little by little, being facilitated by the sweating of the hands. Some have denied this method of poisoning (Blarez).

The makers of lead-pipes suffered formerly more than at present; the improvements in the processes for manufacturing having decreased the risks.

The process for making shot involves a double risk to the workmen, *i.e.*, poisoning by both lead and arsenic. Lead, for shot, is mixed with about five per cent. of arsenic and about four per cent. of antimony. This is done to insure the shot falling "round" instead of elongated or "tailed." In melting and alloying the metals, the workmen are exposed to the fumes; and again in ladling out the melted mass and pouring it through the colander, usually in a lofty tower, they are directly exposed. A new method, by which a rapidly revolving cylinder, with perforated sides, does the work, has been introduced; but the old method is said to give better results.

Type-founders are exposed to the action of lead, type-metal being an alloy of lead, antimony, tin, and copper. Danger arises in mixing the ingredients, in filling the moulds, in trimming and in polishing the type. In the first two stages the fumes of the metal arise; in the latter two stages the dust of lead and the other metals is given off. In recent years machinery has been introduced which obviates so much handling of the metal as was formerly required.

Compositors, or type-setters, are in constant contact with lead in the types, and it might be thought that they would frequently be poisoned with it. They are not specially clean in their persons, and many of them eat doubtless with soiled hands. Cases of lead-poisoning are reported among them, but, according to my observations in Philadelphia, these cases must be rare. I do not recall, among many cases of plumbism from various occupations, having seen one in a type-setter.

Makers of lead-toys, which are made in moulds, are exposed to the same risks as type-founders.

Plumbers are poisoned sometimes with the fumes arising from melted solder, which is composed largely of lead.

Painters, as is well-known, are not infrequently poisoned with lead, which they use for white and for red paint (white and red lead). They often have the symptoms of chronic plumbism, including the contracted kidney, "lead-gout," and optic atrophy. I shall refer presently to the case of a painter who had "pseudo-tabes," due to peripheral neuritis, caused by lead.

Du Mesnil (*Annales d'Hygiène*," 1874, XLI., 25, 335) observed lead-poisoning in workers on lacquered furniture. The lacquer contains lead and is polished, the polishers especially suffering.

Gilders and bronzers, who use lead in some of their work, may suffer from it. Gilders of wood use a varnish containing white lead and litharge.

A rare cause of lead-poisoning is silk weighted with acetate of lead. Dressmakers thus occasionally suffer. A case occurred once in Philadelphia in a seamstress, who was poisoned by biting off the ends of the sewing-silk which had been thus treated. The lead seems to be added to the silk simply for deception, *i.e.*, to increase weight. Alpaca cloth is contaminated with lead; hence tailors working on it may be poisoned (Hime).

Thibault (*Annales d'Hygiène*, 1856, 2 S., tom. 6, p. 55) described a process for tracing designs for embroidering various kinds of materials, which gave rise to some severe cases of lead-poisoning. The design being picked out as a kind of stencil, was applied to the material; then a white-lead powder, mixed with a resinous substance, was made to penetrate the stencil, thus tracing the design; finally a *hot* iron was passed over the design, which, acting on the resinous substance, fixed it. In this process the artisan was exposed to both the fumes and the dust of lead. This industry, which few persons have ever heard of, and which employed in Paris hardly more than a hundred work-people, illustrates the fact that lead is used in the arts probably in numerous ways of which physicians and hygienists hear or know but little.

Lace-making gives rises to lead-poisoning, not in the tedious process itself, as some seem to think, but in a subsequent process for cleaning, or rather for *whitening*, the tissue. In the long slow process of making Brussels lace the thread becomes discolored. It is impossible to clean this by ordinary methods, because of the delicacy of the tissue. It is, therefore, whitened by being pressed between papers in the midst of a fine powder of white lead. Young girls do this work, and often suffer exceedingly (Thibault). In subsequent handling and finishing, the artisan, bending over his task, inspires dust arising from the whitened lace, and this dust permeates the air of the work-room, poisoning others as well as himself.

Lead colors are used for artificial flowers, as red and white lead, and the chromate. They are also used for wall-papers. "Satining" is an especially dangerous process; it is done by friction, and raises a great dust. Making "velvet" paper also is attended with risk (Hime).

Flint-glass contains lead, being chemically a compound of silicon (silicic acid) with an alkaline and an earthy base, the latter being represented by lead. It is this flint, or lead, glass that is used for cut-glass because of its brilliancy. The glass cutter's mill is a revolving disc of wrought or cast iron on which is fed a mixture of sand and water, with which the cutting and polishing are accomplished. In this process, which requires the artisan to bend closely over his mill, dust and fine particles of glass are given off. Emery and putty-powder also are used, the latter containing lead. These are the noxious agents by which these cutters and polishers of flint-glass acquire lead-poisoning.

Beaugrand (*Gazette des Hôpitaux*, 1862, XXXV., p. 7) observed lead-poisoning in workmen employed in vitrifying the enamel labels upon bottles and jars used by chemists and druggists. The enamel contains a vitreous preparation of lead; the lettering and trimming cause a fine dust to arise, which, mingling with the saliva, is swallowed in small but repeated doses. Beaugrand thinks, with reason, that this is the method by which lead dust gets into the system. He says, truly, referring to this little known trade, that the list of industries giving rise to lead-poisoning appears to be interminable.

Archambault (*Archives Générales de Médecine*, Août, 1861, p. 129) some years ago described a curious industry which had for its object to cover an iron surface with a vitreous coating. This was done in making small "crotchets" or hooks for suspending telegraph wires, which were thus insulated. A complex powder, containing ground glass, red lead, and other ingredients, was made to vitrify on the surface of these small hooks. A thick dust was raised in preparing this vitrifying powder, and thus poisoning was caused by the silicate of lead. This preparation of lead, minutely pulverized, can remain suspended in the air for quite a long time. It is readily inspired, and also mixed with the saliva, and with food and drink.

Another minor glass industry has been referred to by Hillairet (*Bulletin de l'Académie de Médecine de Paris*, 1864, XXX., 485), called by the French the manufacture of "verre-mousseline." It has for its object the decorating of window panes, rendering them opaque and adorning them with designs resembling embroidery. An enamel containing oxide of lead is used.

Glazing pots and pans used for kitchen purposes may cause lead-poisoning.

Glazing cards and heavy paper is an industry that may also cause these accidents.

Glazing bricks involves the use of lead and may cause plumbism. In reference to this general subject it should be remembered that glazes and enamels are fusible salts of silicates and borates of potassium, sodium, and lead. Artificial jewels are made from these pastes, which are colored for this purpose.

Napias (*Annales d'Hygiène*, 1883, IX., 3 S., p. 358) called attention to the fact that the makers of brass wind-instruments may suffer with lead-poisoning. This poisoning is caused by working the brass (an alloy of copper and zinc, which always contains some lead); by using solder, in which the proportion of lead varies from $\frac{1}{5}$ to $\frac{1}{2}$; and by certain technical uses of lead for curving the brass.

Snuff and other preparations of tobacco are sometimes adulterated with chromate of lead and red lead. Quite a literature has grown up on the subject (Index-Catalogue, Vol. VII., p. 907). Dr. Owen Rees (*Lancet*, Jan. 21st, 1860, p. 61) has described a case in a snuff user. Poisoning is more likely to occur in the consumer, apparently, than in the workman.

Weavers with the Jacquard loom are exposed to lead from the wearing away of the "lingoes" or leaden weights that are used in large numbers on them. This, according to Hime, is a very considerable risk.

Jewels are sometimes polished on rapidly revolving discs bound with a leaden rim. This is a dangerous process to the workman.

Straw hats are sometimes finished or sized with a preparation containing lead.

File-cutters in former years were often poisoned with lead. This was because the files were cut by hand on a lead anvil. This process raised a lead-dust, which was inhaled and swallowed by the workmen, who leaned closely over their work. According to most authorities this cause for plumbism still exists apparently, especially in England. In this country, however, things are different. Messrs. Disston & Sons, of Philadelphia, extensive saw and file manufacturers, have kindly informed me that the old-fashioned method is not used by them, all their files being cut by machinery, and that instead of using lead they use zinc, which does not wear as does lead. They use quite a number of lead baths, however, for heating purposes, but they have never known of any of their men being affected by the use of these. They state further that from ninety to ninety-five per cent. of the files made in this country are made by machinery and not by hand as formerly in England.

Among the most common sources of lead-poisoning is the manufacture of white lead (plumbic carbonate), red lead (triplumbic tetroxide), and litharge (plumbic monoxide). These are all used as paints.

The ordinary method for making white lead on a commercial scale is as follows: Pieces of sheet lead are supported in earthen jars or crocks, so that they do not fall to the bottom, and into each vessel is poured a small quantity of vinegar. The jars, in great numbers, are stacked up by standing first a layer of jars, covered with boards, and surrounded with spent tan (or horse manure); then on top of this layer a second layer, and so on until quite extensive stacks are built up. The acetic acid attacks the sheet lead, forming an acetate which is at once converted into a carbonate by the carbonic acid that is formed by the fermenting tan. The acetic acid, thus again liberated, attacks other molecules of lead, which in turn are at once converted into carbonate. Thus very little of the acetic acid is required. It seems to act as a carrier for the carbonic acid. After this process is completed the stacks are pulled to pieces, and the carbonate of lead thus formed is removed from the jars, and subsequently washed, ground, and packed. It seems that of all these processes the most dangerous is pulling apart the stacks; in this, a dust is raised which is readily inspired and swallowed.

Litharge, called also *massicot*, is prepared by heating the carbonate to dull redness.

Red lead is made by exposing the monoxide (litharge) for a long while to the air at a very faint red heat (Fownes).

Mr. W. H. Wetherill, of the firm of Wetherill & Bro., of Philadelphia, the oldest firm manufacturing white and red lead in the United States, has kindly written to me as follows: "Most sickness seems to occur from inhaling the white-lead dust, although from the start of melting the lead up to the time of putting it into kegs, as white lead, sickness may occur; and yet the last man to handle the lead, and least exposed, may be the first or only one of a set starting off together, to get sick; the care, the cleanliness, and the constitution of the men having very much to do with it. I have buried two men that were continuously employed for over *fifty years* each, and both continually exposed to influences that sickened and killed many around them, some of whom would be down in a month's exposure, or even shorter time."

Speaking of the red lead, Mr. Wetherill says: "Inhaling the fumes and dust while oxidizing the lead in the furnaces seems to be the cause of lead-sickness in the red-lead department, but a steady run of ten years each for several men, then sickness, then recovery and general good health subsequently, gives weight to the statement that there averages more sickness among farm-hands than among laborers in lead-works."

Grinding the various lead paints may, of course, give rise to poisoning.

Calico-printers, who use some of the lead colors, are exposed to some risk; but the proportion among them of men who thus suffer is probably small.

Finally, it may be of interest to note that Ramazzini hints in his book that the artists Raphael and Correggio suffered with some form of metallic poisoning.

The influence of sex, age, race, climate, and season is not marked in chronic saturnism. Women are said to suffer not so much as men, but this is probably because of their cleaner and more temperate habits. Children are highly susceptible to lead-poisoning as a rule.

All races, in all climates, are apparently equally prone to it. It is claimed by some that this intoxication is more common in summer than in winter, and this because of the moisture of the skin with sweat. There can be no doubt, as Mr. Wetherill has so clearly pointed out in his letter, that idiosyncrasy has something to do with lead-poisoning. Some individuals evidently resist the poison better than others. Thus, the contrast between a man who works continuously for fifty years, and another who goes down in a month, is an illustration of this fact.

The method of absorption of lead has been a subject of some controversy. It is customary to say that it is taken up by the lungs, by the stomach, and by the skin. While not denying that it may gain admission by any of these ways, I am inclined to believe that most of it in all cases of artisans is absorbed from the stomach and bowels. Even in the cases in which it presents itself as a dust it is probably mingled with the saliva, and is thus swallowed in constantly repeated, minute doses all day and even during the night. As to skin-absorption it is by no means proven that it is active. In the cases of painters it seems much more reasonable to suppose that the lead gains admission with the food when the latter is taken with soiled hands.* Lead is not very volatile; the so-called fumes are minute particles, usually of the oxides. As for absorption by the lungs, it can readily be imagined that these minute particles of oxides might be converted in them into some soluble salt and so be absorbed.

The *symptoms* of chronic plumbism are anæmia, a blue line at the border of the gums, colic, in rare cases gastric and precordial pain and vomiting, paralyses of various muscles and muscular groups, especially the extensors of the hands, tremor, cramps, hysterical anæsthesia, a characteristic encephalopathy, convulsions, contracted kidneys, a pseudo-gouty diathesis, and optic atrophy.

The anæmia of chronic lead-poisoning is caused by a diminution in the number of red blood-corpuscles and in the amount of hæmoglobin.

The blue line at the gingival border is rarely absent; in fact, it may often be seen in lead workers who have no other well-marked symptoms.

The colic of chronic plumbism is quite common and characteristic.

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* In this connection, however, it is well to recall the facts collected by Manouvriez (Gazette des Hôpitaux, 1874) under the title of "Local Saturnism." In thirty cases the local effects corresponded to the local application of lead to the skin. Many of these were in workmen. Some had used their feet to stamp lead, and had loss of power in the legs, etc. Anæsthesia has occurred in an area to which lead was applied. The hysterical character of this symptom must not be forgotten.

It is called the "bellaine," and "colica pictonum." The latter name it derives from the fact that it occurred as an epidemic in Poitou, in the seventeenth century, owing to the adulteration of wine with lead. It is also called, expressively, in the West Indies, the dry belly-ache. It is usually a severe, griping pain, centring about the umbilicus, accompanied with obstinate constipation and not relieved by a bowel-movement.

Epigastric and precordial pain, and vomiting, are occasionally seen in lead-poisoning. They are not usually described in the books, and hence may be misleading. I once saw a case in a white-lead maker, in which the pain and oppression almost simulated those of a pericarditis.

The paralyses caused by lead are among its most common symptoms. The most characteristic is the paralysis of the extensors of the hands, producing the well-known wrist-drop. The muscles supplied by the musculo-spiral nerve are the ones to suffer, although curious exceptions for some unknown reason are seen in the cases of the supinator longus and the extensor of the metacarpal bone of the thumb, both of which invariably escape (Fig. 1). The type of paralysis is that of a neuritis. The muscles waste and give the reactions of degeneration. Other muscles that suffer occasionally are the deltoid and some of those of the upper arm. In exceptional cases the legs may be involved, as well as the arms, and then the patient may have either a paraplegia or the symptoms of a more diffused neuritis. Some years ago I described ("Forms of Pseudo-Tabes due to Lead, Alcohol, Diphtheria, etc.," *Medical News*, April 2d, 1892) the case of

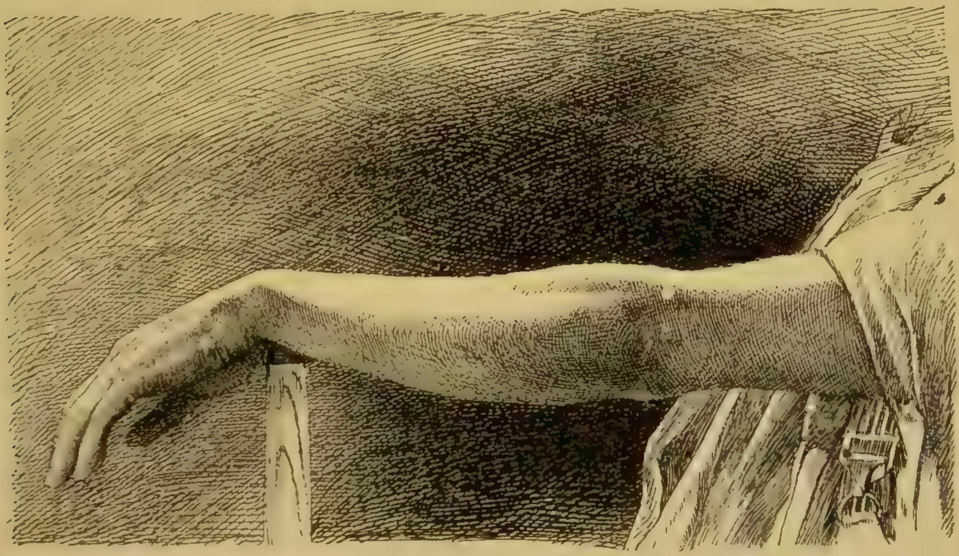

FIG. 1.—Paralysis of the Arm with Preservation of the Supinator Longus Muscle in a Case of Lead-Poisoning.

a painter who presented a "pseudo-tabes" due to lead-poisoning. His case simulated closely one of locomotor ataxia, as he had a markedly ataxic gait. The symptoms were caused evidently by a peripheral neuritis affecting the sensory nerve-endings. The autopsy revealed a perfectly healthy spinal cord, but the nerve-trunks showed some inflammatory changes.

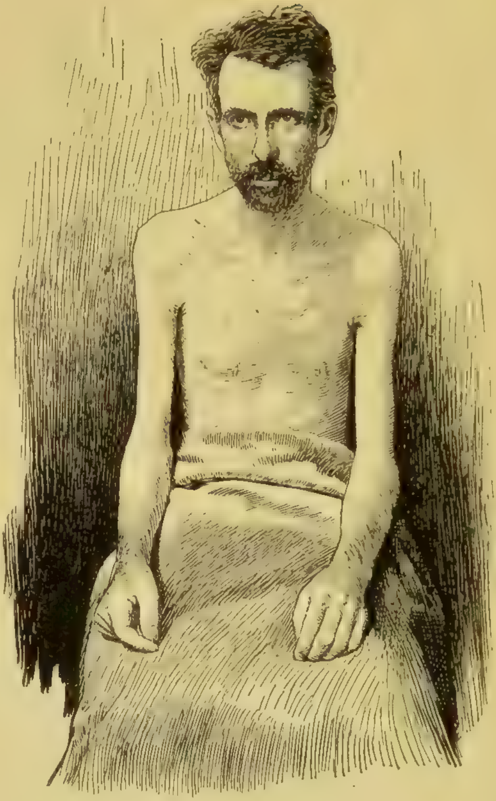

Fig. 2.—A Case of Progressive Muscular Atrophy in a Workman in White-Lead.

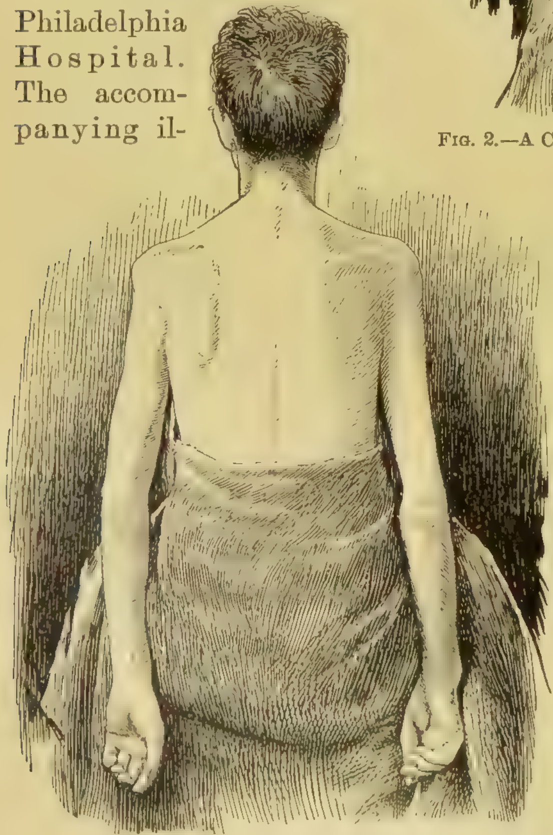

Fig. 3.—Same as Preceding.

Occasionally a type of progressive muscular atrophy, due to long exposure to lead, presents itself. These cases are rare. I have recently seen one such in the Philadelphia Hospital. The accompanying illustrations are from photographs of this patient (Figs. 2 and 3). The man was a workman in a white-lead factory. He has extensive muscular atrophy, involving the muscles of the arms and shoulders, and also, to a less extent, those of the legs. The arms are wellnigh powerless. The muscles have not lost their electro-irritability. Sensation is not involved.

The tremor of lead-poisoning is not so constant a symptom as is that of mercury, yet it is sometimes seen. It presents practically the same type as the mercurial tremor. Beginning as a fine movement in the hands and arms, it increases in amplitude in time. It persists during repose and is often much aggravated by emotion and voluntary movements. It may invade the muscles of the neck, face, and tongue. I have seen it in a house-painter, who was still able to do a good day's work.

Cramps in the affected muscles are seen occasionally.

Anæsthesia of variable extent and location is seen sometimes in lead-poisoning. It closely resembles, and in fact in some cases is identical with, some of the sensory stigmata of hysteria. This subject will be discussed later in a special note on toxic hysteria.

The cerebral symptoms of lead-poisoning are of great interest and importance. They may occur in workmen, as early results of acute poisoning, or they may follow some of the other symptoms in more chronic cases. Delirium and coma are the commonest of these brain states. The delirium may occur rather abruptly, or there may be a slight prodromal stage of uneasiness, sleeplessness, and headache. Sometimes a convulsion is the initial symptom; several of these may occur, the patient persisting in a delirious or passing into a stuporous or even comatose state. In the delirium there are commonly hallucinations of sight and hearing. Tremor sometimes accompanies the delirium, causing the case to resemble one of alcoholic poisoning. Optic neuritis is sometimes seen. The termination is by recovery, by death, or by chronic insanity. In grave cases, with repeated convulsions, like an epileptic status, death is not uncommon. The chronic symptoms, which may endure for life, are melancholia, maniacal episodes, delusions, occasional epileptic attacks, and mental deterioration.

Garrod (Medico-Chir. Trans., LVI., 854) described a form of gout as an effect of chronic plumbism. Since his time it has been rather common to speak and write of saturnine gout. This view has been more generally accepted in England, the place of its origin, than in France and this country. It appears very evident to me that there is some confusion of terms and perhaps of things in this theory. Lead undoubtedly causes arthralgia, a contracted kidney, and a distinct cachexia that resembles gout in a way and may be called a pseudo-gout. But there does not appear to be good and substantial reason for confusing this condition with true gout, which is caused apparently by a distinct poison. The two states are similar, but not identical. The fact that lead diminishes the excretion of uric acid is, however, an argument in favor of it being a promoter of true gout.

Optic atrophy is seen not very uncommonly in lead-cases. Of other ocular nerves the third and the sixth are paralyzed in very rare cases.

The morbid anatomy of lead-poisoning is described in the article on poisoning by lead.

We are more interested here in the *prevention* of lead-poisoning than in the treatment. There is no antidote for lead, when taken in the course of an occupation. A number of substances have played that rôle, and some still enjoy the reputation; but it is doubtful if any of them is infallible. In Derbyshire, among lead-miners and lead-workers, fatty food, especially butter, is considered to be almost a sure antidote to lead-poisoning. Allied to this is the belief in milk. Mr. Wetherill, in his letter, cited above, ascribes importance to the drinking of milk daily. It is possible that it may form an insoluble compound with lead in the stomach. Sulphuric acid lemonade has been much relied upon. It forms an insoluble sulphate of lead, and, from the chemical standpoint, would appear to be efficacious. Experience proves, however, that it will not prevent plumbism. Perhaps one reason is that the men become careless in its use, or come to dislike it.

Proper ventilation is the surest and therefore the best preventive for lead-poisoning in all places where the metal gives rise to either dust or fumes. Mr. Wetherill says: "Owing to arrangements for carrying off the dust, sickness here is so rare nowadays, compared with years gone by, that if one out of fifty workmen gets sick monthly, this number would probably cover it." This statement by an experienced proprietor of extensive lead-works covers the whole subject. The details for accomplishing this ventilation must be left to builders and mill-owners, who understand better what is wanted and how to accomplish it than does the physician unacquainted with technical construction.

Next to ventilation, perhaps even equal to or surpassing it in importance in some trades, comes personal cleanliness. There can be no doubt that lead is frequently introduced into the system from soiled hands in the act of eating. In the case of painters, who are not as a rule exposed to the dust or fumes of lead, this method is probably almost universal. The rule for personal cleanliness, however, applies to all lead-workers. Here, again, Mr. Wetherill's admirable remarks may be quoted. After referring to "cleanliness and common-sense" as the most important means for prevention, he says: "The men who keep well washed, with finger-nails thoroughly cleaned, and avoid the dust when by chance they see it wafted toward them, or wear the respirators provided for them in exposed places, are rarely heard of as lead-sick."

Respirators of various kinds are used in lead works. The difficulty is to get the men to wear them.*

The medicinal treatment for chronic plumbism is largely with iodide of potassium. This salt increases the elimination of lead by the kidneys, in a manner that is not exactly understood. In the acute stage, when the system is probably overloaded with the poison, it is considered best not to use the iodide too soon. It seems to liberate lead from the tissues, and so, it is asserted by some, rather increases the severity of the symptoms. I have not observed this action in any of my own cases. However, it is best to observe the rule by beginning with small doses (3 to 5 grains).

For the constipation Glauber's or Epsom salts are to be given. They act favorably, also, by converting the lead remaining in the intestinal tract into an insoluble sulphate. This action may be increased by adding to each dose a few drops of diluted sulphuric acid.

For the colic an opiate, either a hypodermatic injection of morphine or an opium suppository, may be given. This treatment, by relieving intestinal cramp, may relieve constipation.

For the paralyses, strychnine (preferably under the skin), electricity, and massage are indicated. The galvanic current should be used, because as a rule the faradic irritability of the paralyzed nerves is abolished. The course of the paralyses is sometimes slow in spite of the best treatment. A first attack of wrist-drop usually ends in recovery.

For the encephalopathy and convulsions an expectant plan of treatment is required. The iodides and sulphates should, of course, be used, but much reliance must be placed on a sustaining diet, and simple dilutents, as lithia water, to wash out the kidneys. Lithia salts, which promote the excretion of uric acid, may possibly expedite a cure in that way. They are good in cases also of so-called saturnine gout.

EFFECTS OF PHOSPHORUS.

Phosphorus exists in two forms, *common* and *amorphous* phosphorus. The former alone concerns us here. Common phosphorus is a soft, solid, colorless body, which emits fumes very readily at a temperature of 32°. It oxidizes readily in the air, and is so inflammable that it must be kept under water.

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* In a visit to Wetherill & Bros.' works I saw the respirator in use. It is shaped like a muzzle ("snouts" the men call them) and carries in its end a wet sponge through which the air is inspired. A small valve-like trap rises with each expiration to permit the air to escape. One objection the men have to them is that they interfere with expectoration during tobacco-chewing!

Phosphorus is made almost exclusively from the bones of animals. The bone-ash is subjected to sulphuric acid in weak solution, sulphate of lime and superphosphate of lime resulting. From the latter salt phosphorus is made by a process of reduction with charcoal in retorts under high temperature. The details of this process do not concern us here. The fumes, largely of phosphuretted hydrogen, are burned as they emerge, and the semi-solid phosphorus flows out and is received and solidified under water. The process, therefore, is without danger to the workmen, who, according to Bristowe, do not suffer in the least from phosphorus-poisoning.*

The distillation, however, may be dangerous from the necessity for cleaning out connecting pipes of frozen phosphorus, and from the inflammability of the substance.

One of the minor uses for phosphorus is in the manufacture of a vermin poison. It is made up into a paste with some starchy material to which has been added an oily substance and sugar. There are no fumes generated in this process to affect injuriously the very small number of men engaged in it.

The chief commercial use of phosphorus is for making matches. This is almost as true to-day as it was years ago when Chevallier wrote that 36,000 kilograms of phosphorus were consumed in France annually for match-making, while less than 100 kilograms were employed for all other purposes. Of recent years, however, it has come into use in the tar-color industry; but of any poisonous effects in the workmen from this use of it I have no knowledge.

The phosphorus friction match began to be manufactured in 1833. The industry first became of importance in Vienna, and for many years it had its principal seats in Austria and southern Germany. This accounts for the fact that the earlier papers on chronic phosphorus poisoning appeared in the same region. This poisoning is caused almost exclusively by match-making, comparatively few instances, so far as I know, having been reported as due to other causes. This industry has since spread over the civilized world, and in some countries has great commercial importance. Thus, according to Paton, it is calculated that in the principal European countries from six to ten matches are used for each individual daily.

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* Formerly, however, according to Cazeneuve (Annales d'Hygiène, 1889, xxi., 289) phosphorus-necrosis occurred sometimes in stokers and others engaged in distilling phosphorus. A case had not occurred for twenty years at the time Cazeneuve wrote his paper, and this exemption he thinks due to perfected methods. He refers to cases that had occurred in Lyons. Some of these arose from the old custom of sucking the semi-solid phosphorus into a glass-tube in order to give to it the familiar cylindrical form.

In Great Britain this author estimates that the yearly value of matches made is not less than one and a half million pounds sterling, and that the factories turn out 300,000,000 matches daily. Extensive factories exist also in Sweden and Norway. In the United States the industry is a very large one, the revenue derived from the internal tax on matches alone in a recent year having been more than three millions of dollars. Among large factories are those at Wilmington, Del., and Akron, Ohio.

Not many years elapsed after the introduction of phosphorus matches before cases of chronic poisoning began to be noted. There was at first considerable doubt as to the exact nature and cause of the disease, so little was known of it before. It is thus seen how purely an industrial disease this affection is. The first paper on the subject apparently was by Lorinser, of Vienna, in 1845. He published an account of 22 cases. One of the earlier, and still important, monographs was by v. Bibra and Geist ("Die Krankheiten der Arbeiter in den Phosphorzündholzfabriken." Erlangen, 1847); this was accompanied with an atlas giving realistic pictures of the necrosis of the lower jaw in various stages and of varying extent. Among early cases reported in this country were those of Wood, of New York, and of Hunt and of Boker, of Philadelphia. The most complete paper, in the English language, on phosphorus-poisoning as an industrial disease is by Bristowe (5th Report of the Medical Officer of the Privy Council, 1862). In this paper a detailed description is given of the manufacture of matches, as well as a thorough historical and pathological study of phosphorus-poisoning.*

I am informed by Dr. B. F. R. Clark, on the authority of an old match manufacturer, that both kinds of phosphorus are used for making matches. The red, amorphous kind is used for making "safety" matches. It is not poisonous.

Matches vary in kind, but they depend for their peculiar quality for ready ignition by friction upon a combination of phosphorus and some oxygen-yielding body, usually chlorate of potash, but sometimes nitrate of lead, bichromate of potassium, or peroxide of manganese. The chlorate of potassium causes the rapid ignition, with sharp explosion, that characterizes the most popular matches, which, however, are considered the most dangerous. The proportion of phosphorus in different kinds of matches varies widely. According to Dr. Bristowe, who personally visited many factories, the stages of manufacturing which are especially dangerous are: (1) mixing the composition, during which fumes escape in abundance; (2) dipping, *i.e.*, covering the heads of the matches with the composition, to accomplish which the substance, constantly emitting fumes of phosphorus and phosphoric acid, is spread out on a stone slab; (3) drying, during which process fumes are emitted. Similar processes, according to Dr. Clark, who is quoted above, are still used in this country. In boxing and in some other minor stages fumes are evolved, though in much less abundance. In small factories, however, where all the stages are conducted in one large room, as was formerly the case in some instances in England, all the operatives are exposed to danger. In this country there are said to be very few small factories, most of this industry being concentrated in the hands of a few large corporations, which, it is presumed, conduct the business on a large scale and with improved methods. Certainly, in the United States, in recent years, the number of cases of chronic poisoning reported has not increased, while the industry itself has been growing constantly. Inquiry among surgeons in Philadelphia tends to show that the disease now is a very rare one.*

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* The literature of phosphorus-poisoning has grown too extensive to admit of anything approaching even a notice, much less a digest of it here.—See Index-Catalogue.

Manufacturing chemists use phosphorus for making phosphoric acid, hypophosphite of lime, phosphide of calcium, and the chlorides of phosphorus. According to Bristowe the precautions used by them are effectual for preventing chronic phosphorus-poisoning, which appears to be unknown among their workmen. The operations are conducted under a hood, communicating with a shaft, which carries away any deleterious fumes.

The *symptoms* of the chronic form alone of phosphorus-poisoning will be described here. Persons exposed to the fumes of phosphorus do not suffer apparently with acute poisoning; † on the other hand persons who take phosphorus internally ‡ in poisonous doses do not suffer from its chronic effects on the jaw-bones in cases in which they recover.*

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* Mr. S., who was a manufacturer of matches for twenty-eight years, informs me, through his physician, Dr. B. F. R. Clark, that in all that time he had only two cases of poisoning with phosphorus. One of these cases cost him twenty-five hundred dollars. Both cases occurred in men who had had teeth extracted just previously and who continued at their work without their employer's knowledge.

† An important exception to this rule occurred in a case reported by Fournier and Ollivier (L'Union Médicale, 1868, 3 S., vi., 97). A young woman, who worked in a match factory, had an acute necrosis of one of the maxillary bones, associated with symptoms of acute poisoning such as are observed after the ingestion of phosphorus. Among these symptoms were purpura and cerebral involvement. The case ended fatally in six days after the first manifestations. At the autopsy, however, there was found no fatty degeneration of tissues as is found usually in cases of acute poisoning.

‡ Mr. Simon (Lancet, vol. i., 1850, p. 41) reports the case of a man who appar-

Workers in phosphorus were thought at one time to be affected with bronchitis and even pneumonia as a result of the fumes. In his experiments on animals, by exposing them to dense fumes of phosphorus, von Bibra produced bronchitis, pneumonia, gastric disorder, and "decomposition" of the blood, conditions more resembling an acute poisoning than are seen in workmen, who are not exposed to such dense fumes. He states expressly that in his observations pulmonary disorders did not appear to be common among phosphorus workers. In animals he noted also skin eruptions and falling of the hair, symptoms that are not noted among men. Venereal excitement and abortion are doubtful effects.†

The most characteristic effects of chronic poisoning by phosphorus are in the maxillary bones. The disease is a rather slow, progressive periostitis. The first symptom noted is usually a pain in a tooth resembling, and sometimes mistaken for, an ordinary toothache. It is said to occur only in a tooth previously necrosed. After the tooth has been pulled the wound does not heal, but soon begins to discharge an offensive pus. In time the gum ulcerates and recedes, the alveolus is exposed and begins to exfoliate, sequestra are formed and come away. The disease, in favorable cases, may be checked or cease after a rather limited destruction, but in severe cases it progresses until the teeth, one after another, fall out, the alveolus comes away, and, in the worst cases, large portions, or even the whole, of the maxillary bone (the lower jaw especially) are destroyed. These grave cases may continue for several years and not unfrequently end in death. Constitutional symptoms, of course, mark their progress. The patient emaciates, he has a hectic fever, the sign of septic infection, and he may develop tuberculosis.

Formerly there was some controversy as to the exact method of action of phosphorus in producing these effects. Von Bibra and many

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ently had syphilitic disease in the mouth, who occasionally held a piece of ginger in his mouth. When not thus holding it he carried it in his pocket in contact with phosphorus matches. Extensive necrosis occurred, which was attributed to phosphorus; but the syphilis must not be ignored in connection with this case.

* According to Wegner, if the periosteum of an animal be severely wounded and phosphorus be given in the form of a pill even for months, the periosteal changes do not take place; whereas, if the tibia of a rabbit be partially bared and exposed to the fumes of phosphorus, periostitis is set up similar to that of the jaw (Ringer).

† I am informed by an old match manufacturer that he believes in the aphrodisiac powers of phosphorus, and that it is a common remark among employés that phosphorus has such powers. It is notorious, however, that men are prone to delusional ideas on the subject of aphrodisiasis.

following him held that the action was entirely local; that the poison acted only by way of a decayed tooth. Consequently in a sound mouth phosphorus-necrosis could not occur. In his experiments on rabbits he observed local action on the jaw-bones in cases only in which he had pulled teeth (in one case accidentally breaking the jaw). Billroth, however, whose experience was ample, was confident that the affection could occur in those who had sound teeth, and thought that the only explanation was that phosphorus has a special predilection for the jaws just as mercury has a special tendency to salivate. Bristowe observed that phosphorus-workers frequently had decayed teeth, and yet worked with impunity. He says justly that it must be difficult to determine after poisoning has occurred whether the patient had or had not had decayed teeth.

The lower jaw is more frequently attacked than the upper, and is likely to be more extensively necrosed.

Of Bristowe's 59 cases 44 were in males, 15 in females. These figures are not significant, because we do not know the proportion of males and females employed. The age was generally between eighteen and thirty years. Of the 59 patients 21 were known to have died of the disease, and 25 to have recovered. In 39 cases the lower jaw alone was affected, in 12 the upper alone, in 5 both upper and lower, and in 3 the facts of location were unknown. In one case the whole of the superior maxilla, except the orbital plate, had been lost.

The *pathology*, as already stated, consists in a periostitis and consequent necrosis of bone. In the case of the upper jaw, according to Bristowe, new bone does not form, but in the case of the lower jaw an attempt at the formation of new bone is made, and sometimes successfully.* In one of Bristowe's cases, after ten years, the supplementary bone was large and well formed. In some cases, however, the new bone gradually disappears. According to the studies of von Bibra and Geist the new deposit is richer in organic matter than is true bone.

The *treatment* for phosphorus-necrosis is entirely surgical, and need not be discussed here.

The *prevention* of the disease is to be provided for by proper ventilation and a few other simple precautions. Bristowe gives some striking illustrations of what can be accomplished with proper arrangement of the buildings of a factory and with ventilation. Those stages of the work, during which most fumes are evolved, should be conducted apart. These are the mixing the composition, the dipping, and the drying. The mixing and the dipping, moreover, should be done under hoods, connected with ventilating shafts supplied with a constant draught by a ventilating wheel-fan, such as is used in lead-works. The drying-rooms might be constructed so that it would not be necessary for the workman to enter them, *i.e.*, the frames containing the matches might be slid in.

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* Wegner found that phosphorus in small repeated doses modifies the bones, especially in growing animals. Dense solid bone is formed, which afterward undergoes absorption. This effect, however, does not seem to be noted in children, many of whom have been, and may still be, employed in match-making. The difference is probably due to the fact that they take smaller doses by the fumes.

An experienced manufacturer informs me that he attributes the almost complete immunity enjoyed in his factory to the efficient system of ventilation by which the entire atmosphere of the work-rooms is changed frequently.

Personal cleanliness should be enforced. Workmen should not eat with soiled hands nor in a room in which there are phosphorus fumes. Bathing and change of clothing upon quitting work are recommended by some.

No person should be permitted to work who has carious teeth. Formerly this was prohibited by law in Germany.

In some English factories respirators are worn. These are similar to those worn in lead-works (see foot-note, p. 367), the sponge being wet with a solution of equal parts of fresh slaked lime and sulphate of sodium (Salter).

In England it was the custom formerly in some factories for the work-people to wear on their chests a small open-mouthed bottle or flask containing turpentine. This substance prevents the oxidation of the phosphorus. The plan is of doubtful utility.

Finally the substitution of amorphous phosphorus for common phosphorus, it is said, would do away entirely with all danger. This form gives rise to no fumes. Commercial objections to its use are its greater expense as compared with common phosphorus, and the fact that it forms a highly explosive compound when mixed with chlorate of potassium.

In Germany there is a law regulating ventilation, cleanliness, size of rooms, preparation of the paste, clothing, body-inspection of the workmen, and all important details connected with the manufacture of matches.*

EFFECTS OF COPPER.

Copper is found in the metallic state, as on the shores of Lake Superior; and in various ores, as cuprite (the red oxide), melaconite (the black oxide), malachite (the green carbonate), etc. Copper pyrites is a sulphide of copper and iron; it is from this sulphur ore that most copper is smelted for commercial purposes. Some of the sulphur ores contain also arsenic, mercury, antimony, zinc, and silver, which may be injurious to the health of the men working them.* Sulphurous and sulphuric acid also are products of the oxidation of the sulphur in smelting.

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* This law is quoted by Brouardel, Annales d'Hygiène, 1889, xxi., 207.

Copper is in a high degree malleable and ductile. It melts at about 1,000° C., and the color of the vapor arising from the molten metal indicates the presence in it of copper.

This metal is especially valuable for its alloys, some of which were the earliest metallic substances known to man. Brass is an alloy of copper and zinc; and in addition some varieties contain tin and lead. Bronze is composed of copper and tin, with the addition, according to varieties, of different quantities of zinc and lead. For nickel-plating an alloy of copper, nickel, iron, zinc, and tin is sometimes used.

The evil effects experienced by workers in brass are evidently due partly to the copper which enters so largely into that alloy. Hence the diseases of brass-workers will be considered in this section. It must not be forgotten, however, that brass contains also zinc, tin and even lead, and that some of the bad effects of these metals may be observed. It has been contended, indeed, by some (Greenhow) that the fumes of oxide of zinc cause the peculiar effects seen in brass-workers, but this view is not generally accepted.

Pure, or almost pure, copper is worked by the smelters; also by coppersmiths and artisans who stamp it and who turn and chisel and file it for many purposes, too numerous to mention here. Many objects are made of it, including various utensils and some parts of machinery. I am under the impression, however, that absolutely pure copper is seldom used, the metal being usually alloyed to some extent. In those processes of copper-foundering, in which hammering is the chief method, but little dust or powder of copper is raised. Hence the effects are slight if any. In processes, however, for turning and filing, a fine dust is given off, which displays its cupric constituents when penetrated by a ray of sunlight. This dust may act as an irritant to the bronchial tubes and lungs. It may also be swallowed with the saliva and food, and being changed from its metallic state by the fluids of the stomach, may be absorbed and cause poisoning (Layet). Instances are related of copper turners and cutters being affected with typical gastro-intestinal disorders. Bronzers, who sometimes use a powder of copper, have suffered in the same way.

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* Copper ores are associated with tin-ores. For some of the effects of mining these ores see the section on the "Effects of Tin."

A far more frequent source of poisoning is in the various salts of copper. According to Layet the manufacture of sulphate of copper is not dangerous. Acetate of copper is a rather more dangerous poison when met with in the arts. The manufacture of verdigris is said not to be injurious, as the salt is manipulated in a wet state. It is made largely at Montpellier, in France, by dipping sheets of copper in refuse from wine-vats, the resulting acetate being scraped off. Old copper vessels, utensils, roofing, tubing, etc., acquire a coating of oxide or carbonate of copper; and it is in cleaning and scraping these for the purpose of repairing them or for using the old copper again that workmen not infrequently suffer from poisoning. These processes raise a dust, which being taken into the stomach with the saliva gives characteristic results. Emanations from the ashes taken from furnaces in which copper-ore has been smelted are dangerous, by reason of the oxide of copper with which the fumes are charged. Founders of copper, brass, and bronze objects are exposed to danger from the old sand which they remove and use over again for moulds.

Workers in the various alloys of copper suffer from the combined poisonous effects of the metals. Some specimens of brass have one per cent. of lead in them, and exceptional varieties (as for false jewelry) have even higher. The zinc is usually contaminated with arsenic. Bronzes have even a higher percentage of lead. Some ancient bronze coins of the Roman Empire contain five and six per cent. of it. Modern bronze for statues may contain a fraction of one per cent., while bronze used for locomotive bearings, according to an English authority (Paton), has so much as seven per cent. of this metal. Bell-metal is a bronze, containing, however, some zinc and lead, as a rule. There can be no doubt that much of the evil resulting from brass-working is due to the lead, and, in a minor degree, to the arsenic. Mérat, Christison, Chevallier,* and others of the older observers pointed out these facts. Beside brass-founders, stove-makers, pin-makers, and mechanics on locomotives, have been reported as having been thus poisoned; in fact, many artisans who work in brass suffer in this way. The metal known in France as "white copper" or *tombac* contains about one per cent. of arsenic. It is used for the manufacture of buttons, instruments of precision, etc.;

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* "Memoire sur les Ouvriers qui travaillent le Cuivre et ses Alliages," par Chevallier et Loury, Annales d'Hygiène, 1850, tome 43 et 44. This elaborate paper is a model of its kind. In it the writers aim to demonstrate that there is no true copper-colic.

and those working in it have sometimes shown the arsenical dyscrasia.

Mr. William L. Austin, of The Baldwin Locomotive Works, of Philadelphia, has kindly sent to me, by Mr. Kenneth Rushton, the following information about the constituents and the use of brass in their extensive works. Mr. Willie, their assayer, says, "Alloys of lead and both the brasses and bronzes are made for special use, and it is possible to mix lead with either of these alloys in all proportions. The alloys used in our work contain from eight to ten per cent. of lead. Brasses may contain .005 per cent. of arsenic; a greater percentage than this would detract greatly from their value.

"The workmen in our brass shop and foundry experience no ill effects from their work. If any ill effects were to result from this work, the men in the grinding-room would certainly be the first to complain. We have had a man sitting over an emery wheel, grinding brass castings, for eighteen years, who claims he has not been sick since he was six years old, and who has just been examined for a life insurance policy and accepted."

Mr. Vauclain, the superintendent, endorses the above statement, and adds: "Brass-workmen as a class absorb a very considerable amount of rum; and as the quality varies, so does the health of the brass-workmen."

These statements by responsible observers are in some contrast with those of certain English writers, who regard working in brass as an unhealthy occupation. The better showing of the American firm may be due to better general hygiene, although the prevalence of drinking is to be noted. The large percentage of lead, and the exceedingly small percentage of arsenic in the brass, are also points of interest. As I understand, however, it is in smelting the copper and zinc ores, and in mixing zinc with molten copper to make brass, that fumes containing injurious proportions of arsenic are most likely to be given off.

It must be remembered, too, as said elsewhere, that employers are not always the first to hear of diseases contracted in their employment. Workmen are often reticent in speaking about these affections, from motives which they regard as politic.

Turner (*London Medical Gazette*, 1839, I., 195 and 697) reported a case of poisoning in a young man who was engaged in printing in letters "of gold." The printing was done with yellow ink, and while the impression was still damp the letters were dusted with a powder composed largely of copper. This process may not be used at present; but a somewhat similar one, with various metallic pigments (arsenical, cuprous, etc.), has been used in very recent years in lithographing, and sometimes gave rise to poisoning. I am informed, however, by Avil & Co., of Philadelphia, extensive printers and lithographers, that they no longer use dry pigments, although when they did so formerly they occasionally observed a case of slight poisoning. According to Turner the size used contained lead, and this metal evidently was responsible for some of the effects observed by him. According to Perron clock-makers suffer sometimes from copper-poisoning.

The *symptoms* ascribed to copper-poisoning and to brass-working have given rise to some controversy. The reason for this is in the facts that exposure to pure copper is comparatively rare, and that most sickness arises from the combined effects of the metals forming the various alloys.

Discoloration of the hair and teeth are conspicuous. Ramazzini referred to the fact that these workmen had a discolored complexion and that the beard and hair became green. Instances are recorded of men with light or white hair on whose persons this became so brilliantly green as to make them objects of curiosity. This effect is caused probably by some chemical reaction between the copper dust and the natural oil of the hair; according to Layet the deposit is not merely on, but in, the hair. The teeth may be discolored a greenish blue, more marked about the neck of the tooth and on its lateral aspects. The front teeth are the most stained. According to observations made on copper-workers at Durfort, in France, the bones may show a green discoloration, and so may even the ground in which the bodies of these men are interred (?). It is said also that the urine is green, and discolors the ground where it falls.*

Gastro-intestinal irritation is caused by the swallowing of copper-dust, which probably becomes oxidized and forms salts in the stomach. The symptoms are burning in the throat, loss of appetite, constriction of the chest, nausea, vomiting, epigastric and abdominal pain and soreness, and mucous discharges from the bowels. Men suffering in this way are often anæmic and dyscrasic. Conjunctivitis is occasionally seen.

Irritation of the bronchi and lungs, with muco-purulent expectoration, is caused by the inhalation of copper dust. Fibroid phthisis may result, as from the inhalation of other kinds of dust.

The so-called "brass-founders' ague" occurs in these workmen after long exposure to their work. Layet claims that there is not much that is distinctive in it, but that it occurs also in workers in foundries with other kinds of metal. He attributes it to exhaustion, over-heating, etc. There seems to be no doubt, however, that brass-founders are rather more subject to it than are others.* It is ushered in with a feeling of illness, and consists of a chill with fever, headache, arthralgia, and prostration. Recovery occurs usually in a day or two. There is no periodicity about the affection, as some have claimed. Men who suffer with it are likely to show more chronic symptoms also, as anæmia, weakness, and pulmonary and gastro-intestinal disorders. The possibility of a complication with lead and arsenical poisoning ought not to be ignored. Some observers have attributed this train of symptoms to zinc entirely, but doubtless they are in error. It seems probable to me that this chill and fever are the symptoms of a secondary infection, *i.e.*, an infection possibly by some form of streptococcus occurring at the seat of some bronchial or intestinal lesion that was primarily due to either copper, lead, or arsenic. Oppenheimer (Johns Hopkins Hospital Bulletin, No. 48) reports the case of a bell-founder who had lead-poisoning (colic, blue line, etc.) and who suffered with this ague. The symptoms were prostration, arthralgia, a severe chill, and a profuse sweat. The man worked in the room in which the metals were melted and mixed. The affection was attributed to the inhalation of the white fumes, evidently of zinc. Aldrich examined the precipitate from these fumes and found that they contained zinc oxide and carbonate, but no arsenic.

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* According to Chevallier (*op. cit.*), who is responsible for the statements quoted in the text, the trunks of the trees in the neighborhood of copper-works are colored green with a deposit of the copper-salt from the foundries.

A tolerance for copper and its salts is said by some of the French observers to be attained by those who are long exposed to them, especially if the workmen have had one attack of poisoning. The accuracy of this statement may be questioned.

Nervous disorders have been reported by some as occurring in workers in brass. Hogben refers also to gout and chronic nephritis, diseases which strongly suggest lead-poisoning. Paralysis, tremor, and locomotor ataxia (pseudo-tabes † due to lead?) are also attributed by him to brass. In some of their work, braziers use a solder which is made largely of lead. Hogben does not give lead due credit for all the evil of which it is capable in brass-working.

The *treatment* for poisoning with copper-salts and copper-compounds must be both preventive and curative.

Prevention must be obtained with proper ventilation and avoidance of the dust and fumes. In mixing copper and zinc to make brass, fumes strongly impregnated with copper and with oxide of zinc (as well as with lead) are given off, and to avoid these the workmen cover their mouths with damp cloths (Arlidge). Many of the suggestions made when discussing the effects of the other poisons will hold here. In fact, as we are concerned here, partly at least, with lead and arsenic, it is unnecessary to repeat.

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* Recently official attention has been called once more in England to the condition of brass-founders, and steps urged for investigating and removing the causes of their ill-health. Arlidge says that the trade is an unhealthy one.

† There can be little doubt, I think, as to the identity of these symptoms with those of lead-poisoning. See a paper by the author on "Pseudo-Tabes Due to Lead," Medical News, Philadelphia, April, 1892.

For the injurious effects of copper and its alloys upon the air-passages and upon the gastro-intestinal tract, we have no specific. So long as the causes act the effects will follow. For the lead-colic and other saturnine symptoms the treatment recommended when discussing the effects of lead is of course the proper one. The same may be said of arsenic.

The statements of Pécholier and St. Pierre, that acetate of copper prevents chlorosis, and that those working in this substance do not suffer from this disease, require confirmation; and the immunity from cholera of workers in copper, which has been claimed by Burg,* needs further investigation.

EFFECTS OF ZINC.

Metallic zinc is obtained now for commercial purposes from zinc-blende almost exclusively. It has been obtained in times past from ores containing the carbonate and oxide, but these ores have become scarce.† Zinc-blende contains a sulphide of the metal, and many impurities, among the most important of which, from our special standpoint, are lead and arsenic. According to most authorities, the roasting of zinc-blende may become a nuisance to the neighborhood and prejudicial to the health of the workmen from the evolution of dense fumes of sulphurous acid. Furnaces have recently been constructed, however, in which the sulphurous acid is conducted into chambers in which it is condensed for future use. According to Dittmar, lead impregnates the zinc-vapor in the processes of metallurgy, and arsenic is almost sure to be present in metallic zinc made from blende. These are most important factors in the production of disease. Not a little confusion, and apparently needless controversy, have arisen about the effects of zinc, just as in the case of copper, owing to the fact not having been sufficiently appreciated that some at least of these evil effects are doubtless due to the lead and possibly to the arsenic contained in the ore or in the metal.

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* For a discussion of this subject see Layet's article on "Cuivre (Hygiène)," "Dictionnaire Encyclopédique des Sciences Médicales."

† Various zinc-ores are found and worked in New Jersey, which State is especially rich in them. I am not aware of any systematic study of the effects of zinc mining and smelting upon health having been made in that State. Neither do I know which variety of zinc-ores is most common in New Jersey. The statement in the text is based upon reports of European observers.

Zinc, as already said in the section on copper, is one of the constituents of brass. It is therefore needless to repeat here what was said there about the state of health of brass-workers. From the evidence, I think that neither copper nor zinc is as responsible for the ill health of artisans who work in them as are the lead and arsenic, the former of which is sometimes designedly, the latter usually unavoidably, mixed with them in making brass.*

I think, however, that it may be a mistake to ignore entirely the possible evil effects of copper and zinc. In the process for making the alloy, zinc is added to the molten copper, and fumes of the oxide of zinc are thrown off. Greenhow attributed to these fumes many of the ill effects seen in brass-workers. They undoubtedly contain arsenic in some instances. It must be difficult to estimate accurately the individual effects of poisons in cases in which possibly four of them, as copper, zinc, lead, and arsenic, are acting conjointly. According to Schlockow (*Deutsche Medicinische Wochenschrift*, 1879), the zinc-workers of Silesia suffer with a long train of symptoms, among which it is not difficult to recognize the possible effects of the fumes of sulphurous acid, such as bronchial and gastric disorders, as well as the dyscrasia of lead and arsenic, such as gastric derangement, pallor (apparently anæmia?) amblyopia, pains in the legs and feet, a sense of constriction of the skin, and inability to walk with closed eyes (simulating locomotor ataxia—the *pseudo-tabes* already referred to); also tremor, and paralysis in the upper extremity. Colic and constipation do not occur. After death "chronic inflammatory deposits are found in the anterior and lateral columns of the spinal cord." Arlidge, who quotes the above symptoms from Schlockow, does not appear to recognize the possibility of the evidence of some lead and arsenical poisoning in them.

Iron is protected from rusting by giving to it a coating of zinc—the process of "galvanizing." This is effected by dipping the iron, usually in thin sheets, in a molten bath of zinc, the iron having first been cleaned and prepared with an acid wash. The object of galvanizing is to protect the iron mechanically with a coating of zinc, which undergoes some oxidation by a true galvanic process. In the operations involved the workmen are affected rather by the acid fumes than by the zinc.

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* See the section on "The Effects of Copper."

The *prevention* of bad effects from zinc must be by ventilation, just as was recommended in the section on copper.

EFFECTS OF TIN.

Tin is found in "tin-stone," an oxide of the metal, and as a sulphide. These ores are sometimes mingled; they are associated often with ores of copper, lead, and arsenic. The largest deposit of tin-ores is in Cornwall, England. Tin is found also in the island of Banca, and in Bolivia and Peru.

The tin-mines of Cornwall are very deep, and, according to some reports, are worked at great expense both of money and health. One of the Cornish mines is said to be 280 fathoms deep and to have a temperature at the bottom of 125° F. (Greenhow). These mines are sometimes very wet. The miners suffer from the effects of bad air, dampness and wet, extreme heat, and the physical exertion of climbing out of the mines. According to Bowhay (quoted by Arlidge) the diseases of tin and copper miners are, in order of frequency, bronchitis, emphysema, phthisis, and asthma. These mines are not dusty, because they are so wet. The miners do not appear to suffer with metallic poisoning, although the ores amid which they work are often rich in arsenic as well as in copper and tin.

In extracting tin from the ore the fumes of sulphurous and arsenious acids are given off. The arsenious acid (white arsenic) is conducted into chambers in which it is condensed and recovered for use. In these metallurgical processes the workmen suffer from the sulphurous and arsenical fumes, not from the tin.

Tin itself, in its ordinary commercial uses, is apparently innocuous.

The manufacture of tin-plate is an extensive and important industry, which has long engaged the attention of hygienists.[*] The term "tin-plate" is a misnomer, as the plate is really iron coated with tin. It is from this plate that so many of the ordinary "tin" cups, dishes, and utensils for kitchen use are made. Thin plates of iron are cleaned in an acidulated bath and dipped in molten tin; by this process a thin but durable coating of tin is given to them. In the course of the various processes of this industry the men are exposed to some acid fumes and to great heat. They may suffer some ill effects from these rather than from the tin itself. The remedy is in proper ventilation.

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[*] An early paper, "Sur l'Assainissement des Fabriques de Fer Blanc," by d'Arcet, is in the Annales d'Hygiène, vol. xxviii., 1842, p. 310.

EFFECTS OF CHROMIUM.

Chromium is found in nature as an oxide, usually associated with an oxide of iron, forming the chrome iron-stone. In preparing chrome iron-stone by powdering it, a dust arises, which is irritating to the nose and air-passages (Hirt). Lead chromate is a beautiful mineral, from which chromium was first obtained. Various preparations of chromium are used in the arts.

Chromic acid and the bichromate of potassium are used in calico-printing. The object is to obtain a yellow dye, which is accomplished by first impregnating the tissue with a salt of lead, the oxide of lead being fixed in the cotton by dipping the latter, thus impregnated, into a solution of milk of lime. After this has been done the material is immersed in a solution of bichromate of potassium, the resulting chromate of lead giving it a brilliant yellow color (O'Neill).

Chromate of lead is the brilliant *chrome-yellow* of the painter. In grinding and manipulating this pigment more danger arises from the lead than from the chromium.

Chromic acid and bichromate of potassium are used also for bleaching fatty substances, as tallow and palm oil. The bichromate is used also in the printing of photographs. A very common use for it is in galvanic batteries.

Another pigment is chrome-green (the sesqui-oxide) which is employed in glass and porcelain staining, and in printing bank-notes. Chromate of potassium also enters into the pink color used for earthenware (Butler).

Dyers use the bichromate for mordanting, or fixing, colors. Other compounds, as the nitrate, acetate, sulphate, etc., are used also in calico-printing. The bichromate is used also for mordanting in wool-dyeing, and in dyeing silk and linen.

In chromium works much danger arises from the grinding.

Baer, quoted by Ducatel, refers to a case of fatal poisoning in a workman who, in drawing off with a siphon a solution of bichromate of potassium, accidentally swallowed some of the poison. Although he supposed that he had spit the substance out of his mouth, grave symptoms, as burning in the throat and vomiting of blood and mucus, came on, and he died in five hours.

Duncan many years ago reported the cases of the dyers in Glasgow, who suffered with poisoning by preparations of chromium. They had ulcers and perforating sores, which actually ate their way through the hand or arm. Such extensive ravages have not been reported in late years, probably because greater care is taken to avoid these results.

Ducatel (*Baltimore Medical and Surgical Journal*, Vol. I., p. 44), however, in 1833, said that similar effects were seen in Baltimore where the bichromate was manufactured in large quantities. Painful burrowing and eroding ulcers were produced wherever there was a break in the skin, but, according to Ducatel, not elsewhere. These ulcers were caused by vapors as well as by dust.

Delpech and Hillairet,* in a most elaborate study of the effects of chromium preparations upon the health of the workmen engaged with them, found the most important symptoms to be: 1. Ulcerations of the hands, feet, and, in general, any part of the skin, especially where excoriated beforehand. The action of the chromates is that of an escharotic. These ulcers show themselves naturally by preference on the hands and feet. Violent pain is caused by them. They discharge a sero-purulent fluid, and present some special characteristics, which are described in detail by the writers. Dogs, cats, and rats about these manufactories suffer with similar destructive lesions. Clouet says that rats suffer complete destruction of the soft parts of their paws, which causes denudation of bone. In men these ulcers are sometimes tubercular in form. Various eruptions may occur also about the genitalia. 2. Perforation of the cartilaginous portion of the nasal septum. This is the most characteristic lesion of chromium poisoning. It is claimed by Hime, in fact, that a chromium-worker can be known by his nose. This lesion is to chromium what necrosis of the jaw is to phosphorus. The initiatory symptoms are tickling or irritation of the interior of the nose, then sneezing, more and more frequent and more difficult to control, accompanied with a copious discharge. Epistaxis soon occurs in the majority of cases. A sense of tightness in the nasal fossæ is felt by some, but pain is usually absent. In fact, some workmen do not know that they have a perforation. Purulent coryza sets in with the discharge of hard, brown crusts. The olfactory sense is usually not impaired. 3. Bronchitis and asthmatic attacks occur in some cases, according to certain observers. The writers here quoted do not think that they are general, but that they were due in the cases reported to special causes, not necessarily associated with the chromates. 4. Gmelin recorded cases of cephalalgia, frequently repeated, with slow fever and progressive decline; but his observations do not seem to have been confirmed. 5. Ulcerations, simulating syphilitic lesions, of the tonsils and pharynx, as reported by Heathcote, had not been seen by Delpech and Hillairet. Heathcote's observations, however, seem to be trustworthy.

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* "Etude du Milieu Industrie de la Fabrication des Chromates au point de vue de l'Hygiène," Annales d'Hygiène, 1869, xxxi. A continuation of this study is to be found in the Annales d'Hygiène, 1876, xlv.

When taken internally the chrome salts act as violent irritants. This is so especially of the bichromate of potassium, which promptly causes gastro-intestinal irritation, with vomiting and diarrhœa, and, in severe cases, collapse and death. Internal poisoning is not at all common in the industrial use of chromium preparations, and when it occurs is usually the result of accident.

EFFECTS OF CHLORINE.

Chlorine is a heavy, yellow gas, of a peculiar odor and having a suffocating effect. It is readily soluble in water. It has remarkable bleaching powers, especially for cotton, linen, bones, ivory, and glycerin. It is used for bleaching rags for making paper. The most stable organic coloring principles are instantly decomposed and destroyed by this agent (Fownes). The chief commercial use for chlorine is because of this bleaching quality.

Chlorine for bleaching is used in the form of the well-known bleaching powder, chloride of lime. This bleaching powder is made by exposing pure slaked lime to the action of the chlorine until no more of the gas can be absorbed. Various plans have been adopted for the production of chlorine for the purposes of this manufacture. The most approved method consists in subjecting the oxide of manganese to the action of hydrochloric acid, the results being chloride of manganese, water, and chlorine gas.

The hydrochloric acid necessary for this process was largely obtained formerly from alkali works, from which at one time it used to be allowed to escape, to the damage of animal and vegetable life for long distances around. It is made now from common salt and sulphuric acid expressly for the manufacture of bleaching powder.

In the processes of making bleaching powder the workmen suffer severely with the effects of chlorine gas.

In brick-making, when common salt is used for glazing, chlorine escapes and may constitute a nuisance.

The symptoms caused by chlorine are largely in the throat and respiratory passages, upon which the gas acts as an irritant. The men engaged in removing the bleaching powder after it has been made are obliged to protect their eyes, skin, and mouth, and to work very hard. They become exhausted from the labor and from the insufficient supply of air while they are swathed up and at work in the chambers, and suffer with eruptions and inflammation of the skin, inflamed eyes, bronchitis, asthma, and destruction of the teeth. These men lose flesh and color, and, it is claimed, are liable to phthisis.

Effects of Bromine.

Bromine is a constituent of sea water (24 grains to the gallon) and of some saline springs. The bromine used for commercial purposes is obtained largely from the latter sources. Some celebrated springs of this kind are found in Germany (near Kreuznach, in Prussia); and, in this country, in Pennsylvania and West Virginia. According to one writer, bromine is prepared directly from the waters of the springs in these American States, so much as 125,000 pounds having been extracted in one year. Bromine is found also in Europe in deposits with certain alkaline salts. It is then prepared as a secondary product.

When prepared from the solution (mother liquor) bromine is extracted by the reaction of sulphuric acid and black oxide of manganese upon the potassium salt—a process similar to the one for extracting chlorine. Bromine is liberated as a red vapor, and is conducted into tubes in which it condenses into a red fluid. In this process, and especially in manipulating the fluid afterward, as in pouring it from one vessel into another, the workmen are liable to suffer from its fumes.

The chief industrial use for bromine is for making the bromide salts so much used in medicine. The salts themselves do not cause injury to the workmen, so far as I can ascertain, but the fumes of the bromine are exceedingly irritating and poisonous.

The *symptoms* caused by bromine in workmen are irritation of the respiratory passages, increased flow of saliva and tears, cough, malaise, giddiness, spasm of the glottis, and asphyxia. Bronchial asthma occurs, but chronic intoxication is unknown. The remedy is aqueous vapor. Prevention is sought by the use of cloths or cotton wool to protect the respiratory organs. Men with weak lungs or men who drink intoxicating liquors ought not to be employed.

Effects of Iodine.

Iodine is obtained largely from the ashes of sea-weeds, in the tissues of which it exists in abundance. These ashes, or kelp, are mixed with water and strained. The solution is then evaporated, the common salts, as chloride and carbonate of sodium, being removed as they crystallize. The mother liquor, strong in iodine, is then treated with oxide of manganese and sulphuric acid, as in the process for the extraction of chlorine or bromine, and the resulting fumes of iodine are received and condensed in appropriate receptacles. Iodine is obtained also from Chili saltpetre by a similar process.

Iodine is used in the manufacture of the aniline dyes; also largely for the production of iodide of potassium and iodoform, which are extensively used in medicine and surgery. Some of the iodides are employed also in photography. The industrial use of the iodides is not attended with evil results.

In manufacturing or manipulating iodine care must be observed. The substance occurs in dark scales, and is readily volatilized at ordinary temperatures. It causes symptoms similar to those already described as due to bromine and chlorine. The irritating effects are experienced largely upon the respiratory passages. A few nervous symptoms are observed, especially headache. Iodine is a local irritant to the skin.

Effects of Cyanogen Compounds.

It is but seldom that these exceedingly active poisons exert their power in industrial life. It is said that cyanide of potassium is used in photography, and the old story, now somewhat threadbare, may be recalled of a photographer who absorbed some of this substance through chaps or abrasions on his hands and suffered from the constitutional effects. Such accidents must be of exceptional occurrence. I have not been able to obtain any trace of such mishaps on inquiry among photographers.

Fownes is authority for the statement that cyanide of potassium is used in the processes for gilding and electroplating.

In the processes for manufacturing and manipulating all the cyanogen compounds, especially hydrocyanic acid, it is necessary to use precautions. Anhydrous prussic acid is a dangerous substance even to smell in small quantities.

The *symptoms* of poisoning by prussic acid are fainting, unconsciousness, convulsive movements, paralysis, a peculiar prolonged and forced expiration, and a tendency to speedy death.

For *treatment* there is no specific. Wood recommends atropine hypodermatically as a respiratory stimulant. Artificial respiration and even the injection of ammonia into the veins have been advised.

Effects of Sulphur and Its Oxides.

Sulphur, when manipulated in large quantities, acts as a local irritant to the skin and eyes. Workmen who grind it experience these effects; and in France, according to Bonisson (quoted by Richardson) the laborers who are employed to dust the vines with sulphur against the *oidium* suffer in like manner. The symptoms are conjunctivitis, dryness and desquamation of the skin, loss of appetite, and diarrhœa.

Of far more importance than sulphur itself are its two oxides, sulphur dioxide, SO_2, or sulphurous acid gas, SO_3H_2, and sulphuric acid, SO_4H_2.

The first of these, *sulphur dioxide*, is met with very extensively in industrial pursuits. It is a product of the burning of sulphur in atmospheric air. Consequently, as already shown, it is evolved in smelting various ores in which sulphides of the metals occur. Cinnabar, or the sulphide of mercury, is one of these. In the Spanish mines, as was seen above, sulphurous acid is evolved in great quantities, and is a source of injury to animal and vegetable life. Copper pyrites is a sulphur-ore from which copper is extracted. Sulphurous oxide, as already said, is a product of the oxidation of the sulphur in this ore in smelting. Lead is extracted from galena, which is a sulphide of the metal, and in smelting this sulphurous oxide is thrown off.

It is quite impossible to notice here in detail all the industrial processes in which sulphurous oxide is eliminated. It is formed not only in the burning of sulphur but also in some processes directly from sulphuric acid, the latter yielding up a portion of its oxygen, and sulphurous oxide being set free as a consequence. Hence, in chemical works in which sulphuric acid is extensively used, the fumes of sulphurous oxide are not uncommon.

Sulphurous oxide (or possibly the sulphurous acid which is SO_2 plus H_2O) is a strong bleacher, and is used for bleaching straw in the manufacture of straw hats and bonnets. It is used for bleaching other materials also.

It is met with also in the manufacture of sulphuric acid, alum, and glass; in preparing catgut; in "tinning" sheet iron (see sections on "Tin" and "Zinc"); in preparing hops (Hime). The makers of the old-fashioned sulphur matches are exposed to it. Ramazzini has a reference to matches of this kind, in use in his day, and speaks of the irritating effects of the fumes of sulphur on the respiratory passages. Sulphurous oxide is much used now as a disinfectant. It is produced by burning sulphur in alcohol, the room to be disinfected being tightly closed. Accidents or inconvenience from this use of it are not common. It acts as an irritant to the lungs of those temporarily exposed to it.

Brick-makers are exposed to a combination of sulphurous oxide, carbonic oxide, carbonic acid, carburetted hydrogen, and, in glazing bricks, to chlorine and to lead.

In the case of a laborer in a brick-yard I recently saw gastric symptoms that could be referred only to his work. He was a man, aged thirty-seven, whose work exposed him to the fumes from the brick-kiln. He had constant vomiting, with pain in the epigastric region, and had lost flesh rapidly. There was pain with a sense of fulness also in the lower abdomen. He had complete anorexia, and a constant eructation of gas. The bowels were regular, and the urine normal. He was admitted into the Methodist Hospital, where under appropriate treatment he rapidly recovered.

This case seemed to corroborate the statement of Hirt that sulphurous oxide is more of an irritant to the stomach than to the lungs.

Sulphurous oxide is an irritant to the gastro-intestinal tract and to the respiratory passages; and it exerts a poisonous action upon the nervous system and upon the blood. Hirt claims that it depresses the vagus nerve and the respiratory centre. In animals it causes depression, convulsions, and death (Hime).

The prevention of evil effects is best secured by proper ventilation. According to Hime absorbent media, as water, milk of lime, metallic oxides, as copper and iron, and sawdust; and oxidizing media, as lead dioxide and manganese dioxide, are important means for prevention.

Sulphuric acid, except for its highly corrosive local action when brought in contact with animal tissues, is not a specially dangerous substance in industrial life. It is made from sulphurous oxide (sulphur dioxide), which is obtained by roasting iron pyrites, or copper-ore, or zinc-blende (all sulphur-ores). The sulphurous oxide is conducted into chambers, made of lead, in which it is brought in contact with nitric-acid vapor and water. The nitric acid gives up a portion of its oxygen to the sulphurous oxide, forming sulphuric acid. In this process, which is carried on extensively, the chief inconvenience is from the sulphurous oxide or acid, rather than from the sulphuric acid; and this risk is met with largely during the process of cleaning out the chambers, during which the workmen are exposed to sulphurous acid and to some sulphate of lead. With the precautions, usually observed, of having the furnaces well closed during the distillation, and the chambers well opened and ventilated before cleaning, but little inconvenience is said to be experienced by the workmen. Whatever inconvenience is felt can be obviated by care in these respects. The arsenic contained in some of these ores is given off as arsenious acid, and is separated subsequently from the sulphuric acid, usually by converting it into a chloride. It does not appear to do injury to health in any of these processes.

EFFECTS OF THE OXIDES OF CARBON.

There are two compounds of oxygen and carbon, viz., carbon monoxide and carbon dioxide.

Carbon monoxide, CO, is a colorless, odorless, and tasteless gas, which is produced by the combustion of carbon in an imperfect quantity of oxygen. It is one of the products of the combustion of coal, and burns with a pale blue flame which is so characteristic of a hard coal fire. It is also produced in the burning of illuminating gas; in smelting iron-ores (especially from the smouldering ashes of the same); in burning limestone for the purpose of making lime, and in numerous other instances in which charcoal or coal is burned. It is the deadly gas of the charcoal brazier, which is employed not unfrequently in France by the suicide. Carbon monoxide is also one of the products of the explosion of gunpowder, and its injurious effects may be observed in confined places, as mines, etc.

When carbon monoxide burns it produces carbon dioxide (carbonic acid); practically in fact the two gases are associated in all instances in which they are met with in industrial life.

Many years ago the combustible coal gases, of which carbon monoxide is one, began to be used for fuel instead of the crude coal and charcoal. Guerard ("De L'Emploi Industriel de l'Oxyde de Carbone," etc., *Annales d'Hygiène*, 1843, XXX., 48) discussed the hygienic aspects of this new industrial use of carbon monoxide, and called attention to some of its possible dangers. Already in his time accidents had commenced to occur to the workmen from inspiring the gas. Guerard also relates the case of an aeronaut who, while making an ascent, was overcome by the carbon monoxide, with which and with pure hydrogen his balloon was charged. He was resuscitated after his balloon had descended to earth.

Carbon monoxide is an active poison to animal life. Air charged with four to five per cent. of it may be rapidly fatal to small birds and animals. Even a proportion so low as one per cent., continuously respired, may be most injurious. Ten per cent. is fatal to any form of animal life. In this respect it is in marked contrast with carbonic acid, which can be respired in much stronger percentage (twenty-five to thirty). When the two gases are combined they seem to increase the toxicity of each other, and can do injury in proportions in which singly they would be much less harmful.

Carbon monoxide is a narcotic, not an irritant, poison. Its action seems to be most marked on the nervous system and on the blood. It causes asphyxia by its action apparently upon the respiratory centres and upon the blood direct. Persons who lie down in its fumes, as tramps who fall asleep in the warmth of lime-kilns, and suicides who deliberately lock themselves in rooms with burning charcoal, fall into a deep sleep, which passes into coma and ends in death. The lungs are passively congested. The gastro-intestinal tube is apparently not affected. In the autopsies described by Guerard the appearances just mentioned were noted. The blood is said to be of a bright cherry red in poisoning by this gas and to show some characteristic changes in its spectrum. One of Guerard's cases lingered in a comatose state for thirty-six hours after being removed from the influence of the poison and then died.

Among early symptoms from small doses are giddiness, headache, hyperæsthesia, nausea, and vomiting. In advanced stages of poisoning, convulsions may occur; also slowing of the pulse and respiration.

Carbon monoxide, being imperceptible to the senses, since it is colorless, tasteless, and odorless, is an exceedingly insidious poison. Many instances are on record of persons being overcome by it in totally unexpected ways. It is one of the constituents of "water-gas" (which is made by the action of steam on burning charcoal) and has thus gained access to dwelling houses with fatal results. In its industrial uses its dangers, as already pointed out, are mostly from processes in which it is liberated by the slow and imperfect combustion of coal or charcoal.

The *treatment* consist in removal, of course, from the presence of the poison; artificial respiration; the respiration of oxygen, and sustaining remedies. It would seem that strychnine hypodermatically would be a convenient and efficient remedy.

Carbon dioxide or carbonic acid, CO_2, is also a product of the combustion of carbon in oxygen or in atmospheric air. It may be produced also by the action of an acid upon an alkaline carbonate. It is by the first method, however, that it is usually met with in industrial life, and by which it exerts its poisonous effects. Hence it is commonly produced in the same circumstances as the monoxide; in fact, as already said, the two usually are mixed. It is produced also by the decomposition of animal and vegetable matters, and is met with in volcanic districts, and in the fatal "after-damp" of coal mines (Fownes). It is a colorless gas, with a slight taste and odor. It will not support combustion, and even in a twenty-five-per-cent. mixture with atmospheric air will extinguish a lighted taper.

Carbon dioxide is poisonous to animal life, but not quite so actively so as the monoxide. In overcrowding, being one of the products of respiration, it may cause deleterious effects. Being often mingled with the monoxide it acts under similar circumstances, as about furnaces, lime-kilns, etc. In excavating the ground in some localities it is met with in injurious quantities, as when digging wells, cisterns, or even, it is said, graves.

According to Hime, who presents parallel lists of the symptoms caused by these two gases, carbon dioxide produces dyspnœa, muscular debility, and deep coma; and the heart and lungs are filled with dark blood in contrast with the bright red blood found in the monoxide poisoning. His statement, however, that the monoxide does not as a rule cause coma is not in accord with general observation. It is really difficult to separate the effects of the two poisons. In the experiments of Leblanc upon animals carbonic acid in a thirty-per-cent. mixture with air did not cause serious symptoms, and the animals rapidly recovered after being restored to the atmosphere (quoted by Guerard, *op. cit.*). They were not exposed, however, for a long time. The length of time of exposure is of course an important element in determining serious results. Even a weak solution of the gas, if it continues to act for a long period, may cause grave consequences.

The *treatment* is practically the same as for poisoning by carbon monoxide. Transfusion of blood or the intravenous injection of a saline solution has been suggested.

EFFECTS OF BISULPHIDE OF CARBON.

Bisulphide of carbon, when pure, is a colorless fluid, having a sweet ethereal taste and smell. It is usually mixed with sulphur compounds which give it an offensive odor. Its chemical formula is CS_2. It is readily volatile, highly inflammable, and explosive. It dissolves caoutchouc, and therefore is used in the India-rubber industry. It acts as a solvent also for sulphur, phosphorus, camphor, iodine, oils, and other substances, and therefore is much employed in the arts. Its vapor produces some rather characteristic nervous and gastric disorders.

Bisulphide of carbon is prepared by adding sulphur to carbon which has been raised to a red heat. The sulphur melts and forms a vapor which combines with the carbon, forming an exceedingly volatile compound, which is condensed in a vessel containing ice (Fownes). In the process of making this substance the workmen, according to Gallard, are not exposed to the fumes, and consequently do not suffer with poisoning.

Caoutchouc is a resinous substance held in emulsion in the milky juice of various tropical trees (the *euphorbiaceæ*, *moraceæ*, etc.), the most valuable of which grow in South America. It is modified, and rendered fit for its vast number of commercial uses, by being combined with sulphur—a process discovered by the American inventor, Goodyear. This process is called vulcanization. In order to effect this combination, caoutchouc is dissolved in bisulphide of carbon to which has been added a small quantity of chloride of sulphur. According to early French observers (Delpech, Gallard) who reported the first cases of poisoning by this substance, the mixture consists of bisulphide of carbon 1,000 parts, and chloride of sulphur 2 to 10 parts. In this mixture the caoutchouc is dissolved by the bisulphide and incorporated with the sulphur, which is given up by the chloride of sulphur; and thus vulcanization is effected. The process for vulcanization has probably changed in some ways with time, but the above is the essential action in it. It is not necessary for our purposes here to attempt to follow all the technical minutiæ.

In the process of manufacturing articles from this vulcanized rubber, or of coating textile fabrics with a layer of it (as in making mackintoshes, gum shoes, etc.) the fumes of the bisulphide of carbon are liberated and sometimes affect the workmen.

This substance, according to Delpech, has been used also for extracting the oil from wool; for the extraction of paraffin by distillation from boghead, a bituminous substance, and from coal-tar; for the extraction of various oleaginous substances in the arts; and for extracting bitumen and sulphur from the rocks in which they are sometimes found native. In fact, as said already, sulphide of carbon, because of its property as a solvent of numerous substances besides caoutchouc, as sulphur, phosphorus, camphor, etc., may be used in numerous ways, many of which it is hardly possible even to mention here by name.

Its chief use is in the India-rubber industry. When Delpech first wrote on its poisoning effects it was used extensively in Paris for the manufacture of toy balloons.* These were made in enormous numbers for export. They were made each of two leaves of caoutchouc perfectly adjusted at the edges, which were fastened together when freshly cut by repeated small blows with a wooden mallet on an anvil. The vulcanization was effected by immersing them in a solution of bisulphide of carbon and chloride of sulphur, as above stated. The subsequent processes for drying, inflating with hydrogen, and varnishing were comparatively simple. In the various stages of this manufacture the fumes of the bisulphide of carbon became disseminated through the air and were inhaled by the workmen.

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* Delpech says also that the manufacture of rubber condoms employed many men, and that this industry, like that of the balloons, was responsible for many cases of poisoning with the bisulphide of carbon. He naively adds that these articles were made specially for exportation.

The *symptoms* of poisoning by the bisulphide of carbon are divided by Delpech ("Mémoire sur les accidents que développe chez les ouvriers en caoutchouc l'inhalation du sulfure du carbone, en vapeur." Paris, 1856; also "Industrie du caoutchouc soufflé," *Annales d'Hygiène*, 1863), who first described them, into two stages: first, excitement; second, collapse. In this respect this poisoning is similar to intoxication with ether or with alcohol, a resemblance noted by the early observers. These stages were also noted in experiments on animals; thus Cloez (*Gazette des Hôpitaux*, 1866, p. 358) observed them in the rat and rabbit. A rat, exposed to the fumes from a sponge soaked in bisulphide of carbon, soon became excited and sought to avoid the odor; then his movements were slowed and he had some convulsive shocks; then he fell upon his side, his respirations diminishing little by little until death occurred, in about four minutes from the beginning of the exposure. This observer records the curious fact that in both the rat and the rabbit the right auricle of the heart continued to contract for some hours after the death of the animal, and even, in the case of the rabbit, after removal of the organ from the body.

The first, or stage of excitement, may begin brusquely; it is marked by headache, which the workman attributes to the offensive odor. This headache is sometimes excessive. It may be general or localized, sometimes taking the character of a trifacial neuralgia. Then confusion of vision, vertigo, and even, in some cases, epileptiform attacks supervene. A staggering or hesitating gait is displayed; and muscular pains and formication are experienced. Frequently the sensibility of the skin is exalted (Gallard). The intelligence is excited; the patient is loquacious; sometimes he sings, or he may weep. He is easily irritated, and commits acts of resentment or violence on slight provocation. He is insomnious and restless, or if he sleeps he is troubled with dreams, nightmares, or even a delirium. A true maniacal paroxysm may occur; and an instance of suicide is recorded. The special senses are affected in characteristic ways. The taste is hypersensitive, the appetite exalted; the sense of smell is obscured by a constant subjective odor of the sulphide of carbon; the sight is obscured or diminished—the state known as amblyopia. French observers say that the genital appetite and function are exalted, especially in the male. Women suffer with metrorrhagia, and even abort.

The second, or stage of depression, is marked by a profound weakness, and by a feebleness of intelligence. The patient is depressed, sad, and indifferent to all things. His memory is enfeebled; he finds it difficult to express his thoughts, he cannot find words which he seems to seek. Headache continues. But sensibility shows a marked change; instead of hyperæsthesia there is now a marked anæsthesia and analgesia. The special senses are more involved; amauroses and deafness are present. The genital excitement gives place to indifference and even to impotence. Atrophy of the testicles even has been reported; and in adolescence the development of the gland may be retarded. In women sterility is complete (?), and if by chance they are pregnant they abort. Their breasts atrophy and become painful (Gallard).

It is significant that paralysis, especially a left hemiplegia, was frequently noted in the early cases. This will be referred to again in discussing the hysterical characters of some of these symptoms.

The symptoms, as above described, do not by any means always observe strictly the order and arrangement noted. Atypical cases occur not infrequently.

The *diagnosis* of poisoning with the bisulphide of carbon is usually not difficult. The history of exposure is all sufficient in most cases to determine the cause of the symptoms. There are some special difficulties, however, that may occur. In acute, isolated cases, for instance, especially in those in which the poison has been taken with suicidal intent, there may be some difficulty. Douglass (*Medical Times and Gazette*, 1878, II., 350) records a case of attempted suicide in which the patient, when admitted to hospital, was narcotized, with pale face, blue lips, clammy sweat, frothy mucus, diminished conjunctival sensibility, dilated pupils insensible to light, and stertorous breathing. The man could be roused by repeated urging. He vomited freely. The matters vomited, as well as the urine and fæces in these cases, have a strong smell of the poison. This odor of bisulphide of carbon appears to be one of the best distinguishing points. Chronic cases occur in those persons only who are long exposed to the fumes, therefore probably without exception in workmen only. It cannot then be a matter of difficulty, as a rule, to recognize these cases, which are now so thoroughly described and so well known. Therefore it does not seem necessary to attempt to differentiate this chronic poisoning from such diseases as mania, general paresis, and chronic alcoholism, as some have done.

Toxic Hysteria.—It remains to say a word about the diagnosis of toxic hysteria, not only in cases of poisoning by the bisulphide of carbon, but in those caused by other toxic substances, as lead and mercury especially. The recognition of this complication is often of the first importance both for an estimate of the gravity of the case and for proper treatment. It is a subject upon which we owe most light to the studies of French scientists. Briquet was among the first to note the occurrence of hysterical stigmata in lead-workers. Among seven cases that he reported of hysteria in men, four were in workers in lead. He himself was struck with this coincidence. Later observers noted the same complication and made futile attempts to explain it away. The symptom most frequently observed by them was saturnine anæsthesia. In 1878 Landolt and Oulmont recorded such a case, in which the anæsthesia could be transferred with a magnet. Soon other authors* recorded similar cases, in which saturnine anæsthesia was not only transferable but had even been cured with a magnet. Its hysterical nature was thus clearly indicated. In 1886 Charcot, in a masterly criticism, demonstrated the hysterical character of this and some other symptoms. Debove suggested the term "toxic hysteria" for the hysterical stigmata that are excited by lead and other forms of intoxication, and which often do so much to confuse diagnosis. As Charcot insisted, however, this does not differ from common or "vulgar" hysteria; it is simply the latter, which has for its exciting cause an exposure to some poison. It is most common among artisans. As explained by Letulle, the poisoning does nothing more than prepare the soil for the development of the neurosis in persons predisposed. Guinon places lead in the front rank of these exciting causes of hysteria; but among others are also mercury, alcohol, tobacco, and bisulphide of carbon. Alcohol is of importance because it is a complicating cause, as already shown, in many instances of industrial disease. It may not only aggravate the organic effects of such poisons as lead, mercury, and bisulphide of carbon, but it may greatly augment their hysterogenous powers. Marie (Société Médicale des Hôpitaux, 9 Nov., 1888) has discussed the hysterogenous quality of bisulphide of carbon. An attentive study of the symptoms and cases described in such minutiæ by Delpech and Gallard cannot fail to reveal an hysterical element running through some of them. This is seen especially in such symptoms as cutaneous, mucous, and conjunctival anæsthesia, amblyopia and alterations in the form-fields, and in the convulsive attacks. Letulle ("De l'Hystérie Mercurielle," Société Médicale des Hôpitaux, 12 Août, 1887) has described hysterical symptoms, especially tremor, produced by mercury, particularly in those who work in this substance.

To differentiate the hysterical stigmata successfully from the organic effects of the poison requires an accurate knowledge of these stigmata, which can only be acquired in a special study of hysteria—unfortunately too much neglected in this country. In brief, it may be said that the most common of these stigmata seen in toxæmia are the various forms of anæsthesia (transferrable and even curable with suggestion); tremor (especially in lead and mercurial cases—also sometimes relieved with suggestion), and paroxysmal attacks following the type of four periods, or presenting abortive stages, as described for the classical neurosis.

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* Consult Gilles de la Tourette, "Traité de l'Hystérie," p. 101, for a full discussion of this subject.

The *prevention* of poisoning with the bisulphide of carbon in factories must depend upon cleanliness, change of clothing on leaving work, and avoiding as much as possible the fumes, on the part of the workmen. It was formerly recommended that they should exercise in the open air at the end of the day's work, a suggestion which is not very practicable in the cases of tired workmen. Ventilation is of first importance. As the vapor is heavier than air, arrangements should be made for carrying it off by ventilating shafts opening in the floor or low in the walls. In France it was found that special arrangements, permitting a workman to use his hands by passing them through openings into an enclosure in which the work was done, were ridiculed by the workmen themselves under the name of "magic lanterns," and their use had to be abandoned.

The most important indication in *treatment* is for the removal of the cause; the patient recovers promptly when no longer exposed to the poison. Delpech extolled phosphorus highly for this toxæmia; especially for the sexual weakness. He gave one milligram a day. In one case a carbuncle seemed to cure a paralysis, and this suggested to Gallard the application of a cautery to the neck. Tonics and electricity are adjuvants in treatment.

EFFECTS OF COMPOUND GASES: ILLUMINATING GAS.

Ordinary illuminating gas is the product of the distillation of coal. It is a compound gas, consisting chiefly of hydrogen, the two carburetted hydrogens, CH_4 and C_2H_4, the two oxides of carbon, acetylene, C_2H_2, and small traces of sulphurous acid, sulphuretted hydrogen, etc. When thoroughly purified its combustion is not attended with injurious effects upon health.

From the industrial standpoint this gas does not play an important part in relation to health. In my observation, in fact, although in attendance at hospitals very close to large gas-works, I can recall but little sickness that could be attributed to the injurious effects of this manufacture. I have had men from these works under treatment for rheumatic and other complaints, but not for any affection that could be attributed directly to the poisonous gases. These works usually exhale offensive odors, which can be noticed for long distances. It seems, however, that the more poisonous gases, especially the carbon monoxide, which is one of the combustible, hence illuminating, constituents of coal-gas, are distilled in retorts and do not escape in any large quantities. The lime that is used for removing sulphur, carbon dioxide, etc., acquires a strong odor. Ammoniacal liquor and tar also are products of the manufacture of this gas. Some of these offensive products may act as irritants to the skin and eyes, but do not appear to do so as a rule.

Illuminating gas is an active poison when inhaled, as it sometimes is by accident or design in domestic life. It is narcotic. It is seldom seen, however, in this rôle in the course of its manufacture.

Accidents occasionally happen, however, from the accumulation of this gas in excavations, trenches, and sewers. Instances occur of workmen being overpowered in these situations. Explosions sometimes happen also in such places from the careless use of lights and matches. In Philadelphia recently (April, 1895) a series of violent explosions occurred in a trench which was being dug in one of the streets; these explosions were caused by the ignition of illuminating gas which had escaped from one of the mains and which was fired by one of the workmen, who carelessly threw a lighted match into it.

EFFECTS OF ANILINE.

Aniline is a product of the distillation of coal-tar, which, as we have just seen, is a by-product of the manufacture of illuminating gas. It is formed immediately from nitro-benzene, which in turn comes from benzene, which is a constituent of the tar. The somewhat complicated chemical processes which constitute these various changes need not be described here.

Aniline, when pure, is a thin, oily, colorless liquid of faint vinous odor and aromatic burning taste (Fownes). It is volatile, the greasy spot that it makes on paper soon passing away by evaporation. On exposure to the air the pure oil assumes a reddish-brown color and a resinous consistence, in which form it usually occurs in commerce. Chemically, aniline is one of the aromatic bases; it unites readily with acids, and is remarkable for the great number of crystallizable salts which it thus forms. Its great importance, in fact, comes entirely from the value of these substances, which present all varieties of colors and all tints and shades. The chemical reactions forming these combinations are complex and almost infinite; in fact some of these preparations are proprietary and secret; these reactions, however, do not concern us here.

The immense importance of aniline in industrial life arises from the fact that it furnishes a vast number of dyes and tinctorial agents. The introduction of these has, in the language of one writer, wrought a revolution in the arts and manufactures connected with the dyeing of textile fabrics. Their use, however, is not confined to the textile industries, although they find in them far their largest field; but they are used as stains in other departments of art, a minor one being microscopy. Among the most common of these stains are aniline-purple, or mauve; aniline-red, or rosaniline; aniline-blue; aniline-violet; and aniline-black, or nigrosin. Numerous and, in some instances, fantastic names are used for these substances, as "fuchsin," "magenta," etc.

Aniline, because of its extensive use and its well-known poisonous properties, has now for some years attracted the attention of physicians. Since 1858, when Perkin introduced aniline-purple, or mauve, and thus prepared the way for the rapid development of the aniline industry, numerous papers upon the subject have appeared and not a few cases have been reported of the toxic effects of this substance.

It is important to bear in mind, however, that not all the injurious effects met with in this industry are due to the aniline. In one stage of the manufacture of fuchsin arsenic acid has been used extensively as an oxidizing agent, and some of the bad effects noted were no doubt due to it. Arlidge is authority for the statement that this acid is not used so extensively now for this purpose. The mischief arose from the arsenic acid being allowed to remain in impure specimens. The arsenic having performed its function should be entirely displaced.

Other oxidizing agents used to produce various colors are the oxide and the nitrate of mercury, the peroxide and the nitrate of lead, the chloride of zinc, etc.

Aniline acts as a poison both when inhaled and when taken in the oily form into the stomach. It is by its vapor, however, that it is more often deleterious. Fabrics dyed with its preparations have also sometimes a local irritant effect, but this is probably due to arsenic, and does not so much concern us here in a study limited strictly to its industrial use.*

According to Bergeron (quoted by Beaugrand, "Dictionnaire Encyclopédique des Sciences Médicales," art. "Aniline") this substance given by the mouth to rabbits and dogs causes irritation of the throat and stomach. Clonic and tonic convulsions occur, at first in the hind legs, but soon in the forelegs also; these convulsive shocks are succeeded by paralysis; the eyes are wide open, the pupils dilated; tumultuous action of the heart and embarrassment of respiration occur; sensibility remains intact (although according to other observers it is diminished or even abolished). Shivering occurs, hiccough supervenes, and the animal dies. With large doses violent eclampsia with retraction of the head is caused, and death comes rapidly. According to Ollivier and G. Bergeron the blood is profoundly altered in acute aniline poisoning; it is not coagulable and the corpuscles are deformed and lose a part of their coloring matter. When given by inhalation to animals, shivering and paraplegia, but not such a marked tendency to convulsions, were observed by Ollivier and G. Bergeron. Long-continued inhalation, according to J. Bergeron, produces a true chloro-anæmia with diminution of the red corpuscles and increase in the number of the leucocytes.

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* Instances of the local irritant effects of aniline dyes have been reported by Murray, Medical and Surgical Reporter, 1877, p. 69; by Bailey, Buffalo Medical and Surgical Journal, 1872–73, p. 335; and by Carroll, Philadelphia Medical Times, April 5th, 1873, p. 419.

Among cases of aniline poisoning reported in man may be mentioned those of Knaggs (*Medical Times and Gazette*, 1862, I., 583) and Mackenzie (*Medical Times and Gazette*, 1862, I., 239). In the former a workman broke a vessel containing aniline, and in endeavoring to clear away the débris and the fluid he inhaled the vapor freely. The symptoms were vertigo, headache, and a sense of illness. After some hours, when the physician first saw him, the man appeared to be *in extremis*. His skin was of a blue and livid tint; his lips, tongue, and gums were cadaveric in color; his chest was agitated with convulsive movements. The patient was perfectly conscious, but the pulse was feeble and irregular. The symptoms continued for many hours, and yielded to stimulants, sinapisms, etc. In Mackenzie's case a young man, aged sixteen years, was brought to the hospital in a state of semi-consciousness; the surface of the body was pale and cold; the lips, the mucous membrane of the mouth, the face, and the finger-nails were blue; the pulse was slow and scarcely perceptible. He had vomited and he was conscious of headache and vertigo, of which he complained. He exhaled a strong odor of coal-tar. He had been overpowered while at work in an aniline factory. On the following day the patient still had a bluish tint of the skin and complained of great feebleness; he still exhaled a strong odor of aniline. He recovered gradually under a stimulating treatment.

The *symptoms* of aniline poisoning vary somewhat according to the dose and the duration of exposure. According to Beaugrand (*op. cit.*) the novice at the work is likely to suffer for the first few days with a headache complicated with nausea and vomiting. This is so severe in some cases as to cause the men to quit the work permanently. If the work is persevered in, however, a tolerance is established, and these initial symptoms are not repeated unless the workman happens to inhale an extra large dose of the vapor.

Forced work and the heats of summer are said to induce an attack. In more aggravated cases loss of consciousness follows the vertigo; this in time yields to fresh air, to be followed by an hebetude which may linger for hours, with tenderness of the head. In other instances the workman is seized with a torpor, his face is congested, he staggers and falls like a drunken man in a semi-comatose state; his eyes are turned in, he utters incoherent words, and with difficulty makes some automatic movements. The respiration is difficult and irregular. This state may continue for an hour and even more. In other cases true epileptiform convulsions of the limbs come on, with tetanic spasm of the posterior neck muscles (retrocollic spasm) alternating with accesses of delirium and of trembling. The respiration is irregular, the skin cold and insensible, the face pale; the lips, the tongue, and the extremities are blue; the pupils are dilated, the beatings of the heart frequent and irregular. This state may continue for an hour or more, and subsequently the victim will be extremely fatigued and have headache.

Bronchitis is said by some to be caused by aniline; and chloroanæmia, as observed by Bergeron in animals poisoned with this substance, is often seen in men who work in it. Associated poisoning with hypoazotic acid may cause death—but this does not appear to be a common result in poisoning with aniline alone. In chronic poisoning paralyses are not observed, but areas of anæsthesia and analgesia may sometimes be seen.

In the above list of symptoms no mention is made of hysterical complications, and no observers, so far as I have seen, refer to this subject. It seems probable, however, that hysteria may complicate poisoning with aniline, just as it does that with sulphide of carbon (*q. v.*); and some of the above-mentioned symptoms strongly suggest this possibility.

The precautions to be observed against poisoning are in ventilation and in proper care, by suitably constructed apparatus, to prevent the escape of the vapors.

The remedies consist of removal into fresh air and a stimulating and supporting treatment.

EFFECTS OF NITRO-BENZENE.

This substance is formed by the addition of strong nitric acid to benzene. This is one of the changes in the series leading to aniline, already referred to. Nitro-benzene is a yellow fluid, with the smell of bitter almonds, and has been used as a perfume in the manufacture of soap and in cooking. It is sometimes labelled most improperly "oil of bitter almonds." Chemically it is different from this oil. It is a highly dangerous and insidious poison, numerous instances being recorded of death from its vapor.

Nitro-benzene is used largely for the production of the organic base, aniline, the effects of which have already been described. Hence its poisonous effects may occur in the course of this manufacture. Some observers indeed have ascribed the effects of aniline to nitro-benzene (Letheby), but this seems to be a mistake. It is, apparently, a more active poison than aniline. It is also used, or evolved, in the manufacture of roburite—one of the new explosives. Pockley (*Australasian Medical Gazette*, Vol. XIII., p. 340, quoted in the *Medical News*, Jan. 19th, 1895, p. 76) reports the case of a "mixer" in a roburite factory who had failure of sight, breathlessness aggravated by exertion, blueness of the face, hands, and feet, and vertigo. The patient could count the fingers at a distance of only three yards. There was no central scotoma for red or green, but there was slight contraction of both visual fields. The pupils were dilated and sluggish, the discs pale. Vision improved under strychnine. It appears that breathlessness and blueness of the face are common among these mixers, who are careless of their hands. The symptoms, however, appear to arise largely from the vapor.

The symptoms of poisoning with nitro-benzene are as follows: The eyes are bright, the pupils dilated; the lips and nails are blue; the face is pale, the skin clammy, and the pulse feeble. The mind may be clear for some hours after the poison has been swallowed; then rather suddenly the patient becomes unconscious, with the jaws fixed, the hands clinched and blue, and the muscles rigid. Vomiting occurs in some instances. The breathing is slow. The victim exhales the odor of bitter almonds. Reaction, when it sets in, is slow and may not begin for some hours. In fatal cases death occurs in coma. It seems to be a peculiarity of the poison that it produces its dangerous symptoms after a prodromal period of one or more hours (Taylor, "Treatise on Poisons," p. 635).

When the poison is inhaled, the symptoms, according to Letheby, are similar to those caused when it is swallowed. The cases present the appearance of a slow intoxication, except that the mind is clear until the onset of the fatal coma. This may be sudden, like a fit of apoplexy (Taylor). In one of Letheby's cases four hours elapsed from the inhalation of the vapor to the onset of coma, and this continued five hours until death. It appears from the autopsy in one of Letheby's cases that nitro-benzene is converted partly into aniline in the body, as both of these substances were found in the brain and stomach. In animals the symptoms are somewhat similar to those observed in man. In some cases there is a long initial period of inaction. In animals, however, paralyses and convulsions are apparently more common.

Nitro-benzene is evidently a narcotic poison, somewhat similar to aniline, and presenting some striking resemblances to prussic acid. It is, however, slower in its action than the latter. In cases that recover the after-effects may be long continued, and consist of feebleness and, in some instances, a continued blue tint of the surface, indicating that the blood has undergone some marked change.

There is no specific for this poisoning. The only available remedies are the stomach-pump, in cases in which the poison has been swallowed, and a stimulating treatment. Strychnine under the skin may be used.

Effects of Turpentine.

The only instances of which I have knowledge in which turpentine acts as a poison in industrial life occur in large ship-yards to the painters of the interiors of vessels. Dr. B. F. R. Clark, of Philadelphia, has had experience with cases from large ship-yards in this city, and he is my authority for some exceedingly interesting facts. Dr. Clark says that he is informed that for painting ships the white lead is mixed with turpentine instead of oil. On deck and in the open air this paint can be used with comparative impunity, but beneath decks and in confined spaces the effects upon the painters, it is alleged, are marked and rapid. In the powder magazines, which lie thirty or forty feet below decks, each painter, it is said, works for only five or ten minutes and with a stream of fresh air directed against his face from a hose. He is then taken upon deck where he soon revives from the exhaustion. Remaining below decks too long or repeating the experience too often causes extreme prostration. The symptoms resemble somewhat those of alcoholic intoxication, with, in addition, a coryza, with a watery discharge from the nose and mouth, strangury with bloody urine, and in some cases incontinence. Such attacks, according to Dr. Clark's observations, are common. The painters have learned to expect them, and they have a routine treatment of their own which consists mainly of fresh air and flaxseed tea. These symptoms seem to be due to the inhalation of turpentine,* and are entirely distinct, as may be seen, from those of lead-poisoning, which rarely appear among these painters. Dr. Clark says that he could not find any fatal cases reported.

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* It is but proper to say that in reply to a letter addressed by me to them, the Wm. Cramp & Sons Co., of Philadelphia, stated, first, that painting ships is no more

The following case illustrates this poisoning:

> E. M., æt. 30, has been employed as a ship-painter for several years. He does not use intoxicating liquors habitually, and he has never had lead-poisoning. A few years ago he had an attack similar to the one here reported. One morning recently he went to work as usual. He asserts that he was feeling quite well, but one of his associates thought that he did not appear to be well when he came upon the ship. The work was on deck in the open air. The paint used was white lead, which was mixed with turpentine instead of oil. Shortly after beginning work the man was seized with vertigo and dimness of vision, followed by a slight transient left hemiplegia. There was no loss of consciousness, nor any convulsion. The attack lasted about one-half hour and after it the patient was able to go home unattended.

Dr. Clark has seen other cases, from the same yards, exactly resembling the one just reported. In none of them has he been able to find the blue line, wrist-drop, or any evidence of colic or other symptoms of lead-poisoning. In some of these cases there appears to be a marked mental or emotional element. Dr. Clark says of one of these patients that after the attack "he seemed to be frightened or disturbed and did not return to work the next day."

It is to be noted of these cases that they bear some resemblance to cases poisoned with aniline and nitro-benzene. The effects, however, do not appear to be permanent or serious. The emotional disturbance is not to be lost sight of, nor the possibility, as in poisoning with bisulphide of carbon, of an hysterical element in some of these cases.

The *treatment* with fresh air and flaxseed tea, as instituted by the painters themselves, is probably as good as any that can be recommended.

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deleterious to health than house-painting; and, second, that no serious cases of prostration on account of the fumes of paint had been reported in their yards. They declined to state what the composition of their paints is.

In reference to these statements it may be said, first, that house-painting *is* deleterious to the health of the painters in some instances; and, second, that it is not averred that these cases of prostration are *serious*, but only that they *do* occur. As the Company do not state whether turpentine is used in their paints, the essential point of the inquiry is left untouched in their letter. Therefore the statements in the text, based upon the direct observation of a responsible physician, upon the patients themselves, may be accepted as reliable so far at least as the pathological facts are concerned.

EFFECTS OF THE ALKALIES.

By the term *alkali* is understood in chemistry, and usually in commerce, a hydrate of potassium, sodium, lithium, or ammonium. These substances are caustic in their action, and the first two named are much used in various industries. This is true especially of the sodium preparation—known popularly as caustic soda.

The manufacture of these caustic preparations is attended with some damage to the health of the workmen and also to vegetable life in the neighborhood of the works. This damage results from the acid fumes that escape (especially hydrochloric and sulphurous acids).

Caustic soda, NaHO, is made from the chloride of sodium by practically the same process as that invented by Le Blanc about the time of the French Revolution. The chloride is converted into sulphate by the action of sulphuric acid, and from the sulphate the carbonate is produced by the interaction with the former of limestone in the presence of burning charcoal. It is not necessary to attempt a description of the technical processes for accomplishing this result. In one stage of these processes a mixture of carbonate of sodium and sulphide of calcium results. This mixture is called in the trade "black ash." The carbonate, being readily soluble, may be lixiviated with water, and from it caustic soda may be prepared by adding calcium hydrate as already stated. "Soda ash" and "salt cake"—the latter a sulphate—are various preparations of soda known to commerce. They are results of these processes already described as invented by Le Blanc. An "ammonium process" is now used, which differs in some essential particulars from the old process.

So great a nuisance did the manufacture of alkali become, from the unimpeded escape of the fumes of hydrochloric acid, that a special law, governing this industry, was passed in England in 1863 (known as the Alkali Act). This acid, however, has commercial value for the manufacture of bleaching powder, and for this and for sanitary reasons it is now preserved.

The preparations of soda are active solvents of animal and vegetable tissues. Hence they are used in the paper trade for dissolving out resinous and other matters in the wood and esparto grass used for making paper.

Mr. Grellet Collins, of the firm of Dill & Collins, proprietors of the Delaware Paper Mills in Philadelphia, has kindly given me the following information: "We mix lime and caustic soda ash together, making an alkali liquor, which we use in boiling the wood under a pressure of 100 to 125 pounds for eight hours. We then drain off the liquor which contains the alkali and resinous substance dissolved out of the wood. This black liquor we reduce from 8° gravity to about 30° by a vacuum arrangement and then burn it in a furnace and use the ash over again. The injury is done in the reclaiming and mixing rooms."

The foreman of these mills says that men who have weak lungs cannot work long in either the alkali or "black ash" department. "The fumes from the alkali tanks and the dust from the 'black ash' soon make serious inroads on the lungs when these are not perfectly sound. And it is very trying to the lungs of those who are strong and hearty."

I have no reports of any accurate medical observations on these workmen, but these statements from experienced observers in the mills may be accepted as accurate.

EFFECTS OF QUININE.

Girard noted that workmen employed in stripping off the cinchona bark were subject to a peculiar purulent eruption. Chevallier noted that men engaged in the manufacture of sulphate of quinine were also subject to a cutaneous eruption. Briguet, in his monograph on quinine, noted the same thing, but stated that, as these workmen constantly handled irritating acid and alkaline preparations, the eruption might be due to them rather than to the quinine. Chevallier, in a communication to the French Academy of Sciences, quoted by Bergeron and Proust, reached the following conclusions: The workmen in factories of sulphate of quinine are exposed to a cutaneous eruption which may be of extreme gravity, forcing the victim to suspend or even to abandon his work; that the "quinine fever" described by Zimmer, of Frankfort, as happening among workmen who pulverize the quinine, had not been observed in France; that prophylactic means against the eruption are unknown; that this skin affection is not confined to workmen but may attack those exposed to the emanations from the factories; and that it attacks sober as well as intemperate men.

This whole subject has been ably investigated by Bergeron and Proust (*Annales d'Hygiène*, Tomes 45 et 46), whose conclusions are that an eczematous eruption occurs in workmen employed in quinine factories. But they claim rather inconsistently that this is not a true occupation-disease, but is due to idiosyncrasy; that it recurs very readily; that tolerance to it is only exceptionally established, but that a person who has once had it is extremely liable to have it again; and finally, that this dermatosis is not, as Chevallier claimed, a grave matter.

This eruption in workmen in quinine is eczematous, whereas the eruption caused by the internal administration of the drug is erythematous. It occurs usually on the most exposed parts of the body, as on the hands and forearms, the face, and genitalia (on the latter from handling). Some observers (Layet and Hirt), however, state that it appears also on parts of the body that are covered.

Dr. J. F. Weightman, of Philadelphia, in a letter to Dr. Tracy ("Buck's Hygiene," *loc. cit.*) states that the eruption affects only new workmen, that the majority are affected only once, but that some cannot become accustomed to it; that it is caused by wet preparations, not dry; that it begins on exposed parts of the skin, but may extend to covered parts; that it is characterized by intense itching, followed by an eczematous purulent eruption with swelling of the skin; and that there may be fever. Men with light complexion are more affected than men with dark. These statements are not exactly in accord with those of some foreign observers, but as they are those of a physician who had a direct interest in one of the largest quinine manufactories in the United States, and hence had exceptional opportunities for observation, they are especially worthy of credence.

The affection seems to be rare in the skin clinics of Philadelphia. Dr. Duhring, in a verbal statement to me, said that he had never seen anything distinctive, but that from the description the cases appeared to be instances of a dermatitis due to occupation. A similar eruption, due to the action of an irritant upon the skin, is called by specialists *dermatitis venenata.*

The *treatment* is by removal of the cause, and by emollient applications.

Effects of Tea.

Tea-tasting is a recognized business, the object of which is to judge of the quality of various samples of the leaf. According to Dana (*Medical Record*, 1880, I., p. 85) there are more than one hundred firms engaged in this business in New York City. The process consists in taking into the mouth a small quantity of a freshly prepared infusion of the sample of tea to be determined. Many of these samples are tasted in succession, the expert sitting before a revolving table upon which are arranged tea-cups containing the various kinds and grades of tea. The tea-taster first inhales the aroma and then tastes the tea. As a rule, which appears, however, not to be invariable, the sample is spit out after its quality is estimated. The poorer kinds often are only inhaled. In some instances, according to Dana, a small quantity of the best specimens is swallowed.

An experienced tea-taster, in busy seasons, will sample as many as four or five hundred cups in a day. Dana attempted to estimate the amount of tea, or its active principle, *theine*, that gains admission into the system in an active day's work. Only a small total quantity of the infusion of tea, probably amounting to not more than two or three cupfuls, is actually swallowed. To this must be added the small quantity of theine that is absorbed by the mucous membrane of the mouth, and the volatile oil that is inhaled. On the whole, it does not appear that these tea-tasters, in the course of a day's work, actually take into the system more tea than an ordinary tea-drinker consumes as his or her daily allowance.

It seems evident that some exaggerated ideas have prevailed about the supposed evil effects of this business. Morton (*Journal of Nervous and Mental Disease*, 1879) concluded that the calling is an unhealthy one; but his conclusions have not been generally accepted, and have been rejected, I believe, by the tea-tasters themselves. Dana personally examined a few tea-tasters and found them, almost without exception, unusually robust men. Some of them were in advanced life and had followed the business for more than thirty years. Their testimony, if anything, is rather too favorable and too unanimous.*

According to Eloy (*L'Union Médicale*, 5 Juin, 1886) the excessive use of tea induces a peculiar neurasthenic state, characterized by irritability, insomnia, and a permanent erethism of the nervous system. There are also dyspeptic and cardiac symptoms, as indigestion, with or without gastralgia, and palpitation of the heart.

Slayter (*The Lancet*, April 24th, 1886) reports an interesting case of delirium in a girl caused by the vicious habit of chewing tea. The patient claimed that she had learned this habit when a mill-hand in one of the New England States. Tea leaves, in various stages of mastication and digestion, were discharged from the bowels. Tea-tasters, it is said, sometimes chew the leaves, in which case they probably absorb more of the poison than in tasting an infusion and then spitting it out.

It is possible that tea-tasting may induce chronic tea-poisoning, just as excessive indulgence in the beverage does, but the best evidence seems to be in favor of the view that cases from this cause are neither numerous nor grave.

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* According to Clapton, whose original paper I have not been able to obtain, tea-tasting entails numerous evils. His observations were made in London, and he claims, as quoted by Arlidge, that among the symptoms induced by the business are flatulence, associated with a strong feeling of hunger, which is soon satisfied; peculiar nervous symptoms, as disinclination to go to business, indecision, anxiety, and insomnia; also red and cracked tongue, enlarged cervical glands and coryza. He claims also that these men have small (contracted?) pupils, and that they are out of health, and have a pallid look. Some of the symptoms described by Clapton are evidently due to neurasthenia, and are caused, as he himself admits, as much by overwork and harassment in business as by the tea.

EFFECTS OF ELECTRICITY.*

The vast extension of the uses for electricity in modern life has of course greatly increased the number of injuries and deaths from this agent. These accidents partake somewhat of a surgical character, or at least may be associated with surgical lesions; but in the main they fall, or ought to fall, to the care of the physician, since they depend usually upon internal and even obscure alterations in the tissues for which surgery can give no relief.

The workmen who are particularly liable to be injured by electricity are those who do repairing on the wires of the innumerable trolley and light lines of our cities and towns. Of course other workmen who are employed about dynamos or in any way engaged about electric "plants" may occasionally run risks and receive shocks. It is not necessary, however, to enumerate all these various workmen and their particular employments; they are exposed to practically the same risk, though probably in various degrees, and when they are injured by electric shocks the effects are practically the same in all of them.

With the purely surgical lesions caused by electricity we are not concerned here, except to refer to them briefly. They consist chiefly of burns of the skin and subjacent tissues. When a strong current passes through the human skin for a longer time than is necessary to give a mere shock it is very likely to destroy the tissue with which it is in contact. The longer the duration the greater will probably be the destruction. The hands, of course, are the most liable to be injured, but any part of the body may suffer that is exposed directly to the current. It has been stated that the function of the eye has been destroyed by a strong current, but of any such case I have no authentic information. Whether this could happen without appreciable destruction of tissue may be doubted.

It is extremely difficult to state in scientific terms what is the exact pathology of injury and especially of sudden death from electricity. It is especially difficult to state what exact amount of permanent injury may be left by a shock of electricity in a case in which the shock does not kill. This subject needs more scientific investigation than it has yet received; and it is reasonable to believe that it will be more definitely examined now that so many opportunities are constantly arising. There seems to be an impression that shocks that do not kill do not permanently disable. Yet this does not seem plausible. An agent so powerful that it can kill in an instant should be capable, one would think, of producing intermediate effects—effects that might be permanent, especially in nerve-tissue.

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* Electricity can scarcely be considered an "injurious *substance*," but its consideration seems more appropriate here than elsewhere.

The only exact studies that have been made of the fatal action and effects of electricity in the human body have been made in the State of New York, for the reason, doubtless, that this is the only State in which electricity is used for executing criminals. According to the official report of Drs. Macdonald and Ward (*Medico-Legal Journal*, Vol. IX.), of the execution of four men at Sing Sing, the physiological effects of a fatal current in the human body are as follows: With a current of 1,785 volts, passed through wet sponge electrodes, one of which was fixed on the forehead and temples, the other on the calf of one leg, the heart continued to beat after the first contact of twenty-seven seconds, and a noisy respiration became established after an interval of more than one minute. After a second contact of twenty-six and a quarter seconds respiration had ceased entirely and permanently, and auscultation showed that the heart beat also had ceased. It was believed by the commission that the sudden impact of making the current was the fatal influence rather than the prolonged duration. In a second case, therefore, with the same strength of current three impacts in rapid succession, the contacts each lasting ten seconds, were used. These impacts effectually stopped the respiration, but the heart was still beating so firmly and regularly that it was deemed necessary to turn on the full force of the current again for nineteen seconds, at the end of which time auscultation showed that the heart had permanently ceased beating. After this experience it appeared to be proved conclusively that the duration of the current was an important factor in determining a fatal result (that is, with the strength of current employed). In a third and fourth case the duration of the current was varied slightly, several distinct impacts being secured.

From these observations it appears that with the voltage employed (1,785 volts) the respirations failed before the heart, and that in order to permanently arrest both it was necessary to make and break the current several times and to continue the action of it for from ten to twenty seconds each time. It must be remembered, however, that the officials charged with the execution of these four criminals were interested only in securing the death of the men, and hence may very readily have used more current strength than was necessary to kill. It is evident from reading their report that they took as their guides the heart and respiration and did not consider their duty done until they had secured the permanent arrest of both of these. It does not follow necessarily that the continuance of the action of the heart and respiration for a few seconds or even minutes after the first shock was an evidence that the victims were not dead, or at least would not die. It is probable in fact that the men were killed instantly by the first shock; but this important physiological question is not definitely settled by this report. It is noteworthy that nothing is said in the report about the state of consciousness. Neither does this legal execution determine the minimum fatal dose of electricity. All of these questions, *i.e.*, the minimum dose, the loss of consciousness, the persistence of the action of the heart and respiration even in fatal cases for an appreciable time, and, especially, the infliction of permanent injury in non-fatal cases—all these questions are of great importance in considering the injurious effects of electricity in industrial life. The phenomena will no doubt vary according to the dose.

The anatomical changes caused by a fatal dose of electricity are remarkably slight. The blood is usually found in a fluid state. Van Gieson, in the autopsies on the bodies of the criminals whose cases have been referred to, found this fluid blood, with a dilated and flabby heart, and limited petechiæ on the surface of some organs—notably, one in the fourth ventricle. He reported, however, that the practical results of the microscopic examinations were that the passage of the electric current through the body was attended with no recognizable changes in the tissues or organs. At the points of contact of the electrodes with the skin blistering had taken place from the extreme heating of the water in and around the sponges; this water was estimated to have been raised to the boiling point. It was evidently the cause of the destructive action noted on the cornea and sclera in two of these cases.

The potency of electricity as an hysterogenous agent should never be lost sight of in estimating its effects in industrial life and in medico-legal cases. There can be no doubt that many of the anæsthesias and paralyses left after electric shocks are hysterical in nature. I recently examined a young man who was sueing for damages inflicted by an electric shock received from a low-hanging arc-lamp on the board walk at Atlantic City. While walking under the lamp on a wet day he received a shock from a current which ran down the handle of his umbrella. He fell in a "fit," and when he recovered he had a paralyzed arm. On examination in court it was found that the arm and shoulder were paralyzed completely, hence presenting the type of a *central* paralysis, and that the muscles reacted normally to the faradic current. The blue œdema, so characteristic of hysteria, was present. It was claimed by the plaintiff's lawyers that the nerve- and muscle-tissue had been "disorganized" by the current—an evidently unscientific contention considering what absence of all demonstrable lesion there is after death from electricity, and considering the fact, moreover, that the electrical responses were normal. A verdict was rendered for the plaintiff.

Effects of Dust.

Dust, as can readily be understood, is a result or accompaniment of many occupations. Its injurious effects depend, first, upon its quality, and, second, upon the amount of it which is taken in. In some trades its potency for evil is that of a veritable pestilence, while in others it amounts to little more than an inconvenience. In the former its effects can readily be seen and studied; in the latter many concomitants and other hygienic factors must be taken into account. In the method of study by vital statistics many sources of error and confusion are met with. For instance, according to Dr. Hall, the grinders of Sheffield, whose sufferings formerly from dust could not be doubted, suffered also from bad hygiene, early marriages, and strong drink. On the other hand stone-cutters on some varieties of stone, although exposed constantly to the inhalation of dust, present few symptoms of its evil effects. The subject is so broad, and is involved in various degrees with so many trades, that for the purposes of this article it will be necessary to condense it and limit it in various directions. It will not be possible to give a catalogue and description of every conceivable trade that raises a dust. Only those will be selected in which the effects are conspicuous, characteristic, and unmistakable, and, moreover, of such an aggravated nature as to make them a serious obstacle to the pursuit of the calling. By this method it is believed that, on the principle of the greater including the less, the subject practically can be surveyed, the less important and less injurious trades being referred to incidentally.

Dusts may be divided into two great classes, according as they arise from the inorganic or the organic world. The former may be subdivided into the metallic and the mineral dusts; the latter into the vegetable and the animal. This classification is open to the objection that in some trades the dust generated is a mixture of two or more of these varieties. Thus the grinder's asthma is caused by a dust that is partly metallic and partly mineral; hatters, working on felt, are exposed to a dust that is partly animal and partly metallic and poisonous; spinners and weavers on cotton, flax, etc., are exposed to dust made up largely of vegetable fibres, but these fibres are much more injurious in some instances than in others according to their histological characters, differing from each other more than some of them differ from inorganic dust in their injurious effects. It is necessary, however, for convenience to observe some classification, and the above is adopted as the simplest and the one usually observed by writers on this subject. Some writers (Hirt *et al.*) have attempted to show that the injurious effects depend upon some of the physical qualities of the dust, as for instance, the angularity of the particles. There may be some truth in this. Metallic dust, and dust containing gritty and hard silicious particles, are the most injurious, as will be shown in this article. Only the gravest affections, as already said, will be selected for detailed description, the minor instances being referred to only briefly.

Metallic Dust.

Metallic dust is met with in some kinds of mining, although the dust met with in any particular kind of mining is not necessarily impregnated with the particular metal worked. Most of the dust met with in mining for metals arises from the operations of digging, cutting, and blasting through the rocks and various strata in order to reach the metal. In copper and tin mining, although these ores are likely to contain some of the poisonous metals, as lead, arsenic, and antimony, the miners, it is said, do not suffer with the effects of any of these substances. In lead mining (for galena), however, miners may suffer with lead-poisoning, but whether the metal gains access to their bodies as dust or fumes may be debated. The same may be said of mining for mercury (*q. v.*) which is found as a sulphide (cinnabar). In the great Spanish mercury mines at Almaden the miners suffer with chronic hydrargyrism, but this is caused probably by the vapors of mercury. In estimating the effects of mining upon health it is necessary to take account of many other elements besides dust. Thus the amount of water in the mines is an important factor in producing disease. In some of the Cornish tin and copper mines the water is so abundant that it is necessary to use great engines to keep it constantly pumped out. Of course in such damp mines dust is not much of an evil. The depth, and consequently the heat, of mines is an important factor. Some of the Nevada mines in this country are of great depth, the men being obliged to work in a temperature above 100°. The severity of the labor, even the labor in some instances of merely climbing out of the mines; the constrained positions while at work, the extremes of heat and cold, bad domestic and personal hygiene, and bad habits, all tend to promote pulmonary and cardiac affections in miners which the inhalation of dust may aggravate or which may predispose to the affections caused by dust. More will be said on these general topics in the discussion on the effects of coal-mining.

Electro-platers are exposed to some dust in the processes for polishing, but they do not appear to suffer serious effects from it.

File-cutters, by the old-fashioned process of hand-cutting, were exposed to a fine dust of lead, arising from the leaden anvils on which they cut the files. In America nearly all files are now cut by machinery, as explained to the writer by Messrs. Disston & Co. (see section on "Effects of Lead"), and consequently this evil is avoided.

The trade that is conspicuous above all others for the suffering that it entails from the inhalation of metallic dust is that of the grinder. According to some of the writers of forty and fifty years ago, this trade, as it was then pursued in Sheffield, England, was simply appalling in its evil results. Dr. J. C. Hall, of Sheffield ("The Sheffield Grinders," *British Medical Journal*, 1857), wrote as follows: "If ever there was a trade more poisonous than the fabled valley of the Upas—more slow in its effects, it is true, but equally deadly with the inhaling of the atmosphere of the famous Grotto del Cane—it is that of a fork-grinder who works without employing the effectual means for his preservation which the fan affords." Continuing, he says that over the door of the hull of every fork-grinder, who day after day ventures to grind without the fan, ought to be written: "All hope abandon, ye who labor here"; for his work without this protector, will most certainly kill him.

The best information on the subject of grinder's disease is obtained from Dr. Hall's paper and from a small monograph by Dr. G. Calvart Holland ("Diseases of the Lungs from Mechanical Causes," 1843). Both of these writers were Sheffield practitioners and therefore had unequalled opportunities for observing this affection. Both of them give interesting details of the trade and instructive statistics showing its effects upon mortality. So far as I know their writings are still the best sources for information; because presumably the same conditions exist to-day in cutlery works as they described, except in so far as these conditions have been modified by improved ventilation with fans. In America the subject does not appear to have attracted nearly so much attention as in England, although cutlery is manufactured in this country. I have not been able to find nearly so much written about it here as in England and on the Continent. As the Sheffield writers are the most complete and authoritative, and as the affection in Philadelphia is comparatively rare and therefore has fallen but little under my immediate notice, I shall rely largely upon their writings for the facts stated here.

Grinding edge-tools is of two kinds—wet and dry. The latter method is far the more injurious, as, naturally, it raises far the greater amount of dust. For some tools both methods are used—the mixed method. The tools ground are scythes, saws, table-knives, machine knives, various other kinds of edge-tools, files, pen-knives, razors, scissors, forks, needles, etc. A great difference in their bad effects is observed among the branches of the trade according as the grinders use the wet or dry method and according to the tool ground. The smaller objects are far the most trying and exacting upon health. The worst of all branches are the fork-grinding and needle-grinding. This is because in grinding these small objects the workman is obliged to lean close over the stone and therefore inhales large quantities of the dust, and also because the dry grinding is used.

Grinding was formerly carried on largely in the country by the side of streams which supplied power, but since the use of steam became general the trade has been centred more in large towns, as Sheffield, and therefore the hygienic conditions are not so good as formerly. The men are aggregated in large buildings, divided into rooms, each containing various numbers of wheels. These rooms are called "hulls," the literal meaning of which is *stye*, "and a visit to some of them," says Dr. Hall, "would convince any of my readers that it would indeed be difficult to select a more appropriate appellation." Holland says that a stranger entering one of these rooms would find it difficult to breathe in it.

The dust raised by grinding is composed largely of minute bits of steel. According to Hall, twelve razor-blades forged in the rough, which weigh two pounds and four ounces, lose ten ounces in the process of grinding. This loss represents so much fine metallic dust, mingled of course with dust from the stone. This statement gives some idea of the immense amount of dust produced by dry grinding. According to Holland the concrete masses of this dust formed in needle-grinding have almost the specific gravity of iron.

Needle-grinding is the most destructive among all branches of the grinder's trade. According to Holland young men, aged from seventeen to twenty years, strong and rough from the plough, who engaged in this work in his day, were, as a rule, dead before they were thirty years old. This frightful mortality was in spite of the fact that they worked but six hours a day. The average age of these grinders, according to Holland's statistics, was only 25¾ years. Of all those who died of whom record could be obtained only two were over forty years old. One man, aged thirty-six years, told Dr. Hall that he was a "very old man" for his trade.

Fork-grinding also is a most destructive branch of the trade. "Such is the destructive tendency of this branch," says Holland, "that grinders in other departments frequently refuse to work in the same room." Only about twenty per cent. of the men engaged were above forty years of age, and several of these had commenced the work late in life or had worked intermittently. More than one-half of the men employed were under thirty years of age. Among 20 deaths 17 occurred under forty years of age. These figures sufficiently attest the frightful mortality among the workmen in this branch.

Dr. Holland's book, which is rich in statistics, reviews all departments of this melancholy trade, but the facts and figures quoted above are sufficient for our purposes here. The grinding of the larger and heavier tools, such as scythes, saws, etc., which is done largely by the wet method, is not nearly so destructive to health and life as grinding small objects. In recent years the wet method of grinding is superseding to some extent the dry method for small objects, and this doubtless will effect a decided improvement in the mortality rate. Even when Hall wrote his paper (in 1857) this change was being effected and the consequent improvement was becoming notable. According to his statistics the avearge age at death of grinders working under as favorable circumstances as possible was about forty years, which was a distinct gain over the rates noted by Holland. It must be said, however, that these statistics do not appear to have distinguished sufficiently the various branches of the trade.

The *symptoms* of grinder's disease are those of a slowly advancing bronchitis with asthma and sometimes with emphysema; later dilated bronchi with excavation are observed; then, consolidation, and breaking down of lung tissue occur as the terminal processes. The exact relationship of these processes to a tubercular infection in grinders is an interesting and important subject, which, of course, did not receive much light from the earlier writers. They endeavored, in fact, to draw distinctions between true "consumption" and grinder's "asthma," but the state of pathological knowledge in their day was not such as to permit them to do otherwise. It is quite evident from reading their reports of cases that they had to do with a disease that was essentially tubercular, but it would be interesting to have the exact relationship of this infection to the grinder's unwholesome occupation investigated by the methods of modern bacteriology. I do not know whether this has ever been done. There can be no doubt that the influence of the grinder's trade is that of a predisposing cause. The inhalation of the irritating particles of steel into the lungs causes a constant catarrhal bronchitis and pneumonitis, with increase of connective tissue; and this prepares the soil for a tubercular infection which characterizes the latter stages of the disease. Hence to describe the disease in all its details is to describe the various stages of a more or less chronic bronchitis passing into a destructive tubercular process. It is scarcely necessary to do this here.

Dr. Hall says that the disease usually begins with some digestive disorder. The breathing becomes short and difficult even on slight exertion. The complexion becomes a dirty white. The countenance is indicative of distress. The patient has a sense of constriction across the chest and a sense of dryness in the back of the throat. To these succeeds a cough, at first dry, but later accompanied with expectoration, which is in the beginning frothy. This expectoration sometimes contains dark-colored matter, which has been attributed to the carbon from smoke in the air; but the addition of nitric acid causes it to disappear, indicating that it is not carbon, but probably pigment matter. Under the microscope cells containing pigment, sarcinæ, elastic fibres, pus and mucus cells, and silicious and metallic particles are found. According to this observer the general symptoms are not in proportion to the extent of the disease in the lungs. According to his reported cases, however, the usual symptoms of pulmonary tuberculosis, as loss of weight, cough, expectoration, hæmoptysis, and the usual physical signs, are easily recognizable (*op. cit.*).

It is scarcely worth while to enter into a discussion here as to whether inhaled particles of dust can become imbedded in lung tissue. The subject has excited some controversy, in which eminent pathologists have engaged. Virchow contended against this idea, the theory being that the ciliated epithelium kept the offending matter swept out of the bronchi. The practical observations made upon grinders, miners, granite and other stone-cutters, and potters, all of whom are exposed to most injurious varieties of dusts, and exhibit an abnormally high death-rate from pulmonary diseases, support the belief in the injurious effects of these substances when inhaled. The observations of Greenhow upon miners' lungs (*Transactions of the Pathological Society of London*, 1869),* of Arlidge upon lung disease from the inhalation of dust (*British and Foreign Medico-Chirurgical Review*, 1875), of Gandner and Coates upon the lesions in a potter's lung, of Alison upon the disease of the stone-cutters of Edinburgh, as well as of numerous French and German observers, all tend to support the view that in whatever way the inhaled particles act, they succeed in laying the foundation for pulmonary phthisis. Even admitting that the black masses expectorated are not masses of carbon or metallic substances, but that they are altered hæmatin of the blood, this after all is a strictly secondary question, the vital, or rather mortal, fact being that the cause (dust inhalation) acts constantly to produce an effect (pulmonary phthisis). Into these profound pathological questions we cannot enter here. Statistics prove beyond a doubt that dust kills. Thus, taking deaths in male adults in England from all causes as 1,000, it is seen that of these 220 are from phthisis, while among earthenware manufacturers (potters, etc., who are exposed to a most irritant dust) 473 deaths from phthisis occur, and among Cornish miners (who are exposed to a peculiarly irritant dust, but also to other contributory causes) the deaths from phthisis amount to 690 (Nottu). Finally, it may be said that analysis has proved the existence of foreign substances in the lungs of persons exposed to dust. Gregory, of Edinburgh (quoted by Hall), found that small black concretions in the lungs of a collier yielded carburetted hydrogen and other usual products. The specific gravity of some of these masses in the lungs of grinders has indicated the presence of iron.

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*Greenhow found bronchial catarrh, interstitial proliferation, adherent and thickened pleura, increased density of lung, areas of consolidation, nodules, dilated bronchial tubes, and diseased glands. A fluid like India ink could be squeezed out of the lung tissue, and this left on evaporation a black residue which was insoluble in boiling nitric acid. He believes in the extraneous origin of the pigment, and the appearance of the lung which he depicts is conclusive on this point. Zenker, it will be recalled, found a red pigment in the lungs after inhalation of the red oxide of iron.

The post-mortem appearances are those of chronic bronchitis, with dilated bronchi, enlarged bronchial glands, adherent pleura, cavities, consolidation, and in some instances dilated heart and granular kidneys. Indulgence in alcoholic drinks probably contributes to the kidney and cardiac changes.

For the *prevention* of grinders' disease Hall makes many suggestions. The brutish indifference of many of these English workmen passes comprehension. They appeared formerly to accept their fates as inevitable, and were not only indifferent but even hostile to methods devised for their relief. Some of them declined to use the fan, and they even opposed its introduction for fear that a diminution of the high death-rate would lead to a reduction of wages! Dr. Chadwick says that when he and others at Sheffield once tried to get grinders to use methods for prevention the men said that they preferred a high death-rate with high wages. They even introduced their very young children as apprentices to their nefarious trade before the factory laws regulated the matter. When business was good they thus increased their earnings, which they often spent in dissipation. The one effectual means for preventing high mortality is the use of ventilating fans and tubes to carry off the dust. Of late years these have been extensively introduced and with admirable effects. The fan should be required by law. The substitution of wet for dry grinding should be made when possible, and the two processes ought not be carried on in the same room, as it is senseless to expose wet-grinders to the dust of dry-grinding. Finally the hours of labor should be regulated, and children of tender years should not be employed, since they are very susceptible to the bad effects of the dust, and by laying the foundation of the disease at an early age they contribute to the high mortality in early adult life.

The *treatment* for the disease is that which is adapted to chronic bronchitis and tuberculosis. But it is needless to say that, so long as the calling is pursued, this treatment has but little controlling effect.

Mineral Dust.

Among those who are exposed to mineral dust to an especially injurious extent are miners, potters, glass-cutters, and stone-cutters. In some other trades mineral dust is encountered, but those mentioned furnish a type, a description of which answers more or less exactly for all. Diamond and gem cutters are exposed to an irritating dust. Diamond cutting, as practised in some parts of the Low Countries, is especially injurious.

An extensive literature has grown up about the diseases of miners, and especially about the so-called "miners' phthisis." This affection after all, just as in the case of the grinders, is only pulmonary tuberculosis. Therefore there is no very good reason for describing it apart from that affection. In former years, before the bacillus of tubercle had been discovered, and consequently before the infectious origin of "consumption" was known or dreamed of, it was customary to attempt to draw distinctions between the various kinds of phthisis. Thus grinders' asthma and miners' phthisis were looked upon by some as distinct morbid entities. These views no longer hold. All these destructive lung disorders are now known to be tubercular, and to arise from infection by a recognizable bacillus. The inhalation of dust is simply a predisposing cause. The more irritating the dust the more active and persistent will be the consequent bronchial catarrh, which forms the nidus for the bacillus. Hence it is seen that in some dusty occupations the tendency to pulmonary tuberculosis is more marked than in others. This is owing in part to the nature of the dust inhaled. Metallic dust, of which the substance is exceedingly hard and of which the points and edges are sharp, is of course one of the worst. Steel is especially mortal, as has just been shown in the case of grinders. Silicious dust, which is encountered by potters, is also exceedingly injurious, as is also the dust from some, but not all, kinds of stone. In the cases of miners the quality of the dust varies greatly according to the substance mined and the strata in which it is worked. The Cornish mines are said to be especially harmful, although they are very damp. Coal mining, which raises a dust that is really vegetable in origin but that acts practically like a mineral, excites many cases of phthisis. It is absolutely essential, however, to recall, as urged in an earlier part of this paper, that many factors must be taken into account in estimating the injurious effects of occupation upon health. In the cases of all miners this is especially true. In the case of the coal miner it is necessary to consider that he is exposed not only to dust but to injurious gases, to deprivation of sunlight, to severe toil in a constrained attitude, to exhausted air, and, not unfrequently, to the effects of bad habits and poor domestic hygiene. All these various factors differ widely in different mines and in different localities and countries, and they present a complex subject which cannot be much more than stated here. As far as pulmonary infection by the bacillus of tubercle is concerned, however, it may be said that they all predispose to it and favor its spread. The fibroid form of phthisis, in which there is overgrowth of connective tissue, seems to be not uncommon in miners, and this fact is probably due to the persisting irritating effects of the dust inhaled.

The coal miners of Pennsylvania furnish undoubtedly a quota of consumptives, but how large this quota is in proportion to the population, and how high the death-rate from tubercle is among them I have no means of ascertaining. It seems that a careful study of their vital statistics has never been made, as has been done in England by Greenhow for the Cornish miners. Dr. Fussell, of the University Hospital in Philadelphia, tells me that he sees in that institution occasional cases of phthisis in miners from the coal regions, but that they are simply cases of tuberculosis and have nothing special in them.

It is proper to say that some authorities still claim that a true "anthracosis,"* as distinct from tuberculosis, may exist. This anthracosis is characterized by an infiltration of carbonaceous matter in the lung tissue with fibroid proliferation. Arlidge * holds to this view. The presence of coal-dust is claimed by Layet to have been observed in other tissues, as in the mesenteric glands; and Soyka (quoted by Arlidge) detected it in the liver, spleen, and kidneys. In some instances the lung looks as if "steeped in ink." A black expectoration exists in some cases, and this may persist for a long time after the work has been abandoned. Rindfleisch claimed that he had been able to recognize the structure of coniferous wood, completely carbonized, in a small particle removed from the lung of a dust-inhaler. It is probable that soft coal is more injurious, as being more dusty, than hard coal. Ogle maintains that true tuberculosis among miners is comparatively infrequent, and that coal dust has a specific action against tubercle. But this seems to be an extravagant assertion, which may be traced to the fact that this observer insists upon a distinction between tuberculosis and the lung disease in miners. A bacteriological examination ought to settle this disputed point. That the lungs of miners contain a large quantity of carbonaceous matter does not alter the fact that the destructive process may be due to the action of the bacillus of tubercle.

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* As specimens of word-coinage I may give here the terms devised by Zenker for the various forms of phthisis due to dust. Pneumonoconiosis (*πνεύμων*, a lung, and *κόνις*, dust) is a generic name for the whole class. Anthracosis (*ἄνθραξ*, coal) is the form due to coal-dust; siderosis (*σίδηρος*, iron) that due to iron. Chalicosis (*χάλιξ*, gravel), byssinosis (*βύσσος*, cotton), and tobaccosis are other special designations.

The *symptoms* of miners' phthisis are those practically of phthisis from all causes. The disease is sometimes chronic, especially when it assumes the fibroid type. It may be preceded by a slow bronchitis, steadily progressive or marked by exacerbations. Dyspnœa is often extreme in this stage of bronchitis. Cough and expectoration are more or less constant. In later stages, when tubercular infection has become well established, the usual symptoms of progressive breaking down of lung-tissue, with emaciation and failing strength, are seen.

The *treatment* is that for chronic bronchitis and pulmonary tuberculosis as seen under all circumstances.

In conclusion it may be said that miners suffer with other complaints, the most common of which are anæmia, rheumatism, and nystagmus. The latter is described elsewhere.

Stone-cutters and quarry-men suffer in various degrees from the inhalation of dust. The extent of the evil in their cases depends upon, first, the character of the dust and, second, the circumstances amid which the work is pursued. Quarry-men are liable to accidents and to the effects of very hard labor. If they work under ground or in deep quarries they may suffer with the effects of dampness. The quality of the stone has much to do with the extent of pulmonary disease among stone-cutters. Some stone is much more dusty than others. A sedimentary stone, for instance, that was formed originally simply by the deposit of earthy and silicious particles under water, is much more liable to give off a large quantity of dust than is a stone that was fused in early geological ages—the igneous stones, for instance, like granite. Although the particles from these latter stones are exceedingly hard, yet there is not much true dust, only the particles actually displaced by the contact with the tool being thrown off. These probably do not carry far in the air, and are mostly too large to gain access to the alveoli as dust. This was the explanation given by Hamilton, of Aberdeen (quoted by Arlidge), for the fact that the masons and polishers at the Aberdeen quarries do not suffer much if any with industrial phthisis.

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* Arlidge (*op. cit.*) has an extended discussion of this whole question, with citation of many authorities.

Slate-quarrying, mill-stone working, and working in light sandstone produce bronchial irritation, interstitial proliferation, consolidation of lung tissue, tubercular infection, and death. Edinburgh stone, that produced from Craigleith, has long been known to be especially injurious to the lungs of those working on it. This effect was noted early in this century, and was the object of study by Professor Alison. His statements are most pessimistic, and his views were possibly somewhat exaggerated. It is probable that the lung disease was induced to some extent by other factors, as exposure, heredity, and bad habits. Still the fact remains that a large mortality from pulmonary phthisis was noted among these men.

The pathology is practically the same as has already been noted. Peacock (*Medico-Chirurgical Review*, 1860) in a case of a mill-stone worker noted the post-mortem changes of tuberculosis, as is evident now from reading his description. These were tubercular masses, consolidation of lung-tissue, cavities, and diseased bronchial glands. After combustion of dense portions of the lung silicious particles were found in the ash in larger quantities than are found in normal lung-tissue.

In stone-cutters, as in grinders, lesions of the eyes occur from flying pieces of metallic or mineral substances.

Among workers in mineral dust potters rank among the foremost of those who suffer from its inhalation. In fact, according to Hirt's table, one class of these workmen—flint-grinders—are at the very head of the list for their mortality rate. To understand the reasons for, and the circumstances of, this exposure it is necessary to enter briefly into a study of some of the details of the trade. Clay, of which substance all pottery is made, in the natural state is an hydrated silicate of alumina. It is the most widely disseminated mineral substance in nature. When mixed properly with water clay can readily be moulded into the innumerable shapes that it assumes in pottery. When, being thus moulded, it is baked, it undergoes a chemical change, the water being driven off and the anhydrous silicate of alumina being left as the finished product. For decorative purposes various coloring matters are used; these are laid on before the piece is "fired," *i.e.*, baked, and become incorporated with the substance of the clay, or are held to its surface with some vitreous material, or flux, which forms thus a glazed surface on the object. Various metallic substances are used for the pigments, and lead enters largely into the flux. According to the statement of the foreman of some large potteries near Philadelphia, communicated to me by Dr. Summer, this flux is composed of litharge (lead oxide), clay and flint. Clay thus baked is one of the most durable products of human skill; it does not wear out or undergo organic change in its substance from the action of air or water (or if any it is very slight), the objects made from it being mostly destroyed by breakage alone. In some forms, it will absorb water, but it will not again become truly hydrated.

Clay is found in several varieties, and for practical purposes has to be mixed with other substances. It is sometimes mixed with or made up of ground flint. Flint itself is a silicious substance, being a variety of quartz. One variety of clay, much used for the manufacture of porcelain, is a product of the decomposition of feldspar and therefore is found usually in association with granite. It is called *kaolin*, a Chinese name, as this variety of clay was first used in China, and formerly was exported from that country. It is this clay of which the fine Chinese and Japanese porcelain is made. It is now, however, found in abundance in other countries, as in Nebraska in the United States; in Saxony, where it is the substance of the famous Dresden china; and in England, where it is mined and used in large quantities. Kaolin has as a basis a crystalline substance which, as in other clays, is an hydrated silicate of alumina. In mining kaolin, which has various local names, and in grinding all varieties of clay, which has to be done with great care and thoroughness, much dust is raised. This dust, being almost entirely silicious or of *glassy* consistence, is exceedingly irritating to the lungs, ranking next to metallic dust in its power for evil, if indeed it does not even surpass it.

It is not necessary for us to enter further into the details of the potter's trade. It is an industry of great antiquity and of almost universal dissemination among primitive peoples. It is now carried on in every civilized country. It is sufficient to say that in addition to the grinding of the clay, the baked product is subjected sometimes to another grinding process for polishing and for removing inequalities from the surface. This is done on lathes or with sand paper, and raises clouds of dust. A prolific source of dust, according to Arlidge, is this process for scouring, or cleaning the surface of the pottery after it has been baked. Dr. Warman ("Diseases of Potters," Report of the State Board of Health of New Jersey, 1887, XI., 97) says that the scourers are exposed to a terribly irritating dust and that they are the chief sufferers. They are mostly women, and thirteen years is the longest that any one of them had worked in the Trenton potteries.

The diseases of potters, incident to their trade, have long attracted attention. Among them is lead-poisoning, which has already been referred to (see "Effects of Lead"). The subject that interests us here is the involvement of the lungs from the inhalation of dust.

Among English writers Arlidge, who practised in the Staffordshire pottery district, has written with the most attention to details ("Diseases Prevalent Among Potters," London, 1872, also his larger work, already quoted). According to his statistical table bronchitis and phthisis were the most common diseases in male potters. Bronchitis was twice as common as in the general population. Among American authors Dr. Warman, in the valuable paper just cited, has written on the subject. His observations were made in the extensive potteries at Trenton, N. J., in which five thousand men, women, and children are employed. He gives an admirable description of the trade as carried on in this important centre. The workpeople suffer with phthisis, lead-poisoning, rheumatism, digestive disorders, and anæmia. The scourers, as already said, suffer most from dust, but workmen in many other branches also breathe a dust-laden air. Much dust is raised from the dried clay; when in a moist state the clay is carelessly thrown about, and becoming dry is ground under foot. Ill health is aggravated by unclean habits, bad ventilation, and intemperance.

The symptoms of potter's lung disease are not specially distinctive. They are simply those of chronic bronchitis and asthma, passing into tuberculosis. Arlidge says that dyspnœa is extreme and out of proportion to the bronchitis present. From his description of the lesions, however, this discrepancy does not appear to be striking. He is one of those who endeavor to draw a distinction between "potters' asthma" and ordinary pulmonary tuberculosis. We are sure, however, that this distinction will not hold under the critical study of modern pathology. The long-continued bronchitis leads to some fibrosis, but in the end the lung tissue breaks down under the ravages of the tubercle bacillus, and cavities, adherent pleura, and diseased bronchial glands are found after death.

Greenhow ("Third Report of the Medical Officer of Privy Council," 1860) has calculated the mortality-rate from pulmonary affections for the potters of Stoke-upon-Trent. He found a death-rate from these affections for male inhabitants of 7.21 per 1,000, as compared with a rate of from 2.97 to 4.46 in selected healthy rural districts; while for females the death rate was almost equally high; and more than half of these deaths in Stoke were in potters, although potters form only about one-third of the population of the place. This shows an exceedingly high death-rate from lung disease for potters, and points to a special operating cause. According to Ogle's table ("Report of the Registrar-General," 1885, Supplement, p. 26) the comparative mortality figure for earthenware manufacturers is among the highest on the list of occupations.*

Powdered china clay (the same apparently as kaolin) is used for sizing cotton thread preparatory to weaving it. The object for this, according to Buchanan ("On Sizing Processes in Cotton Manufacture," etc., "Report of the Local Government Board," 1872),† is to give tenacity to the thread and to prevent friction during its passage through the machine. Formerly flour and tallow were used, but later the china clay was added to avoid discoloration and to aid the weaving. Later it was used by the English as a mere adulteration to increase the weight of the cloth. The first incentive to this fraud was the cotton famine caused by the American Civil War. Sometimes a deliquescent salt is used in addition. In weaving it is necessary to keep the air of the mills warm and moist in order that the sized thread may be properly manipulated. In spite of the moisture, however, much dust is given off from the sizing, and constitutes a source of disease. It conduces to pulmonary affections, just as it does when encountered in other trades. Cotton-weaving predisposes to these affections also because of the high temperature and artificial moisture, without ventilation, that are maintained in the mills. The work thus becomes very exhausting, inducing anæmia, gastric disorders, and bronchial catarrh. Buchanan's investigations were made at the request of the workmen, who in this instance showed an intelligent appreciation of the demands of health.

Organic Dust.

Dusts from the vegetable and animal worlds are sometimes the sources of disease. The dusts that are classed thus, however, are not always purely vegetable or animal, as the case may be, but often contain a great deal of mere dirt, which is probably mostly earthy, hence mineral.

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* It is 1,742, as compared with 556 for clergymen, 599 for gardeners, 631 for farmers, on the one hand, and 2,205 for inn servants, and 1,839 for Cornish miners, on the other.

† An abstract of this paper is in the Medical Times and Gazette, 1872, i., 633.

The two vegetable substances that are conspicuous for raising an irritating dust are cotton and flax.

Cotton gives off a dust especially in the early stages of its manufacture. These early stages are opening the compressed bales, picking out and sorting the cotton, and passing it through various machines, such as "scutching" and "carding" machines, the objects of which are to free the cotton from impurities.* It is thus converted into a fluffy mass which is adapted to spinning. The dust of cotton, or "flue" as it is called, is composed largely of cotton-fibre, and it has an appreciable taste and smell, especially to a person unused to it. It causes irritation to the nose, throat, larynx, and bronchial tubes. The symptoms are dryness of the throat, huskiness of the voice, cough, and expectoration. The cough is said to be worse in the morning, the expectoration being tenacious and difficult to be gotten rid of. Fibres of cotton have been found in the sputa with the microscope. In time proliferation of connective tissue ensues, as in most cases of industrial phthisis, and dyspnœa and emaciation occur. In many cases it is evident that a true tubercular infection takes place.

Dr. F. F. Simpson, of Spartansburg, S. C., in a letter to Dr. H. W. Cattell, which the latter has communicated to me, describes an affection of the tonsils in cotton-operatives in that State due to their custom of sucking cotton fibre strongly into their mouths in some process of their work. These fibres become encysted in the crypts of the tonsils, thus becoming a source of constant irritation, and giving rise to what in that section is not inaptly termed "weavers' tonsils."

According to Greenhow's statistics (*op. cit.*) the annual number of deaths from pulmonary diseases in one of the cotton districts of England was 9.75 for each 1,000 males and 9.28 for each 1,000 females—a high proportion. According to Ogle's table the comparative mortality figure for cotton and linen manufacturers is 1,088, which is rather high.†

Flax gives rise to a dust that is even more irritating than that from cotton. Dr. C. D. Purdon, who observed its effects among the workmen in the linen factories in and around Belfast, has described them in a short paper ("The Diseases Which Prevail Among Workers in Flax," *Dublin Journal of the Medical Sciences*, Vol. LXII.). The flax-dust is called "pouce," and "it is produced when the fibre is cleaned by machinery from the decayed wood and earth that had adhered to it in the steeping pools." In the process of "scutching" this dust is given off in large quantities. Fortunately the cleaning process takes place during only a comparatively brief period of each year, that immediately following the gathering of the flax. But flax-dressers are exposed all the year round. The symptoms described by Purdon are dryness of the throat, cough, and dyspnœa, due to bronchitis, paroxysms of distressing cough, with straining and vomiting, and even expectoration of blood-stained sputa; and, finally, in many cases, a true tubercular phthisis. According to this writer "six per mill" is the proportion of deaths per year from phthisis, a proportion of which we have no means of judging except from his statement that it is high.

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* The statement in the text is sufficient for our purposes. The minute description of machinery and the use of technical terms, in which some writers indulge, only confuse this subject for most readers.

† It is not so high, however, as that for medical men (1,122), musicians (1,314), and law clerks (1,151), all of whom, one would suppose, had a more healthful occupation than a cotton worker.

According to Vallin (article "Rouissage," "Dictionnaire Encyclopédique des Sciences Médicales") the process called in French "rouissage," in English "water-rotting," or "retting," and which has for its object liberating the fibre of flax and other textile plants from the colloidal matter that agglutinates them, is disagreeable and incommoding rather than really unhealthful. The putrid waters resulting from the process are not likely to give rise to disease from their being drunk, because the foul smell is an effectual safeguard against this mistake. Flax-workers suffer also from various skin eruptions, especially a "perifolliculitis" due to irritating materials, oils, etc., met with in their work (Leloir). Greenhow's statistical study (*op. cit.*) shows a high mortality from pulmonary affections in districts in which flax-working is carried on. The workers in jute and hemp are exposed to some of the same evils as afflict those who work in flax.

Among other kinds of vegetable dust that may do injury to health is that from wood and from grain and flour. Wood-dust varies widely in its character, as can readily be understood. Some kinds are much harder and more irritating than others. Carpenters and cabinet-makers are exposed to wood-dust, but it is doubtful whether they suffer from lung diseases as a consequence. Workmen in lead-pencil factories, who saw the grooves in the cedar handles for the reception of the lead, are exposed to dust, and according to Merkel * suffer frequently from consumption. Workmen employed on mad-der, Peruvian bark, and chicory also suffer to some extent. The threshing of grain and especially the grinding of it are dusty occupations. Hirt makes rather a poor showing for millers. He says that of every 100 of them who are sick 42 are suffering from pulmonary complaints, and nearly eleven per cent. from consumption. According to Ogle's table their average mortality figure is 957.

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* A translation of Merkel's work will be found in The Medical Record, vols. ix. and x. Merkel discusses the pathology and morbid anatomy of dust inhalation, but uses Hirt's statistics very freely.

Among dusts of animal origin may be named, first, that from wool. Weavers and cloth shearers are exposed to it, but it does not appear to be so injurious as that from flax or even cotton. As in the case of the latter, however, the weavers are subjected to bad hygiene in the weaving-rooms. Women working on sewing-machines on woollen fabrics, and makers of shoddy, are said to suffer from pulmonary diseases as effects of their occupation. A bronchial catarrh with febrile reaction is known as "shoddy fever." In Lyons a belief is prevalent that silk workers are especially prone to tuberculosis, and Givre (*op. cit.*) has made a statistical study of the subject. It is probable, however, that this fact, if true, may be due to the bad habits of the workmen and especially to the bad hygiene of their lives. Hatters, as already shown, are subject to a dust from fur, which, however, is prejudicial rather because it is impregnated with nitrate of mercury than from any inherent quality in itself. Workmen on horn, bone, and ivory are exposed to dust, especially when the substance is sawed, but exact figures are wanting as to the injurious effects. Merkel relates the case of a turner of horn in whose lungs post-mortem were found particles of horn-dust.

According to Hirt's statistical table (*op. cit.*, p. 30), showing the rate of prevalence of phthisis in the total number of sick among workmen who inhale dust, needle-grinders and file-cutters are at the head of the list of those exposed to metallic dust.* Flint workers lead on the list of those who inhale mineral dust; cigar-makers those who inhale vegetable dust (but this list does not include flax-dressers), and brush-makers those who inhale animal dust. But the figures for many of the trades on his list show that the inhalation of dust is insignificant in its effects upon the workmen in those particular occupations. This table of Hirt's, in fact, is open to criticism in several particulars. The number of cases observed by him was evidently small. With reference to brushmakers it is said that they inhale minute particles from the bristles. These arise from the clipping and trimming. They are extremely irritating to the lungs.

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* It must be recalled that file-cutters by the old hand-methods were exposed to lead-poisoning, but it seems that in some processes they are exposed to the evils of grinding—a distinct process from cutting. Hirt's figure for them is probably not too high.

Among those exposed to animal dust in minor degrees, and who need only be mentioned here, are hair-workers, upholsterers, fell-mongers, and button-makers. Some of these are exposed to a specific infection which will be described in the next section.

EFFECTS OF THE ANTHRAX BACILLUS.

This bacillus, the cause of malignant pustule and of wool-sorter's disease, is a rod-shaped organism, usually several times longer than the diameter of a red blood corpuscle. It grows in chains, each individual bacillus being adherent to the next in the series by the end, and thus forms dense felted masses, or mycelium. It has been studied with great care by Davaine, Koch, Pasteur, and others, and its life-history and pathological effects are now quite well understood.

The clinical fact had been known for many years that workmen in certain wools and hair, especially those from Russia and South America, were subject occasionally to a local, highly malignant sore, attended in time with symptoms of systemic infection, and not unfrequently proving fatal. The specific cause of this malignant sore was discovered by Pollender in 1849, and is now known to be the anthrax bacillus. Only in recent years, however, have several clinical varieties of anthrax infection been recognized; and as most of these are pre-eminently industrial diseases, they interest us especially here. Wool-sorters, and other workmen in wool and hair in England, especially in Bradford, noted, after the introduction of alpaca, mohair, and camel's hair, the occasional occurrence of symptoms indicating the action of a poison, which symptoms were not associated with or dependent upon a local sore. These symptoms set in insidiously and made rapid progress. The most prominent of them were a sense of illness and weakness, with pain in the chest and an oppression in breathing. Moist râles and pleuritic effusion were observed. The temperature was not raised as a rule, and chills and sweating were not present (Arlidge).* The cases sometimes were rapidly fatal, nineteen hours being the limits to the duration in two cases. This affection was recognized as a form of anthrax poisoning by Dr. Bell, of Bradford, who first investigated it.

It is thus seen that the anthrax disease may be divided into two classes, the external and the internal.

The *external* form, or the malignant pustule, has been observed in most quarters of the globe, and is the commonest. In America it has not been so prevalent in an epidemic form as in Europe, yet it has been observed. A remarkable series of cases were reported in 1871 by Dr. A. H. Nichols ("Report of the Board of Health of Massachusetts," 1870–71, p. 86) as occurring in one factory in Walpole, Mass., during a period of three years. Twenty-six cases occurred in all, and it is noteworthy that at least ten of these were of the internal variety of poisoning. The workmen affected were engaged in the manufacture of curled hair. This hair was imported from Buenos Ayres, where it was sheared from the necks and tails of living wild horses. At the factory it was picked apart, after being taken from the bales, and then sorted and passed through a picking machine, which separated all foreign substances. It was next spun into ropes, boiled and finally dried in a heated compartment by which the curl was set. During these processes, according to Dr. Nichols, the hands of the workmen were constantly brought in contact with the hair, while in the vicinity of the picking machine the air was filled with dust, composed largely of dried animal matter. This hair was originally, and perhaps is still, imported into New York, whence it was distributed to the factories in New England, Philadelphia, and Baltimore. This instance serves as an illustration of the devastation that can be wrought by this bacillus. Of the 26 cases 14 were fatal. Many other instances have been reported, especially in England. Malignant pustule begins as a vesicle, the result of direct inoculation, usually on some exposed surface, as the hands or face. The vesicle breaks down into an ulcerated sore; an extensive areola, densely swollen and deeply injected, spreads wider and wider; and the veins, lymphatics, and neighboring glands become involved. The symptoms of septic infection set in, and in fatal cases the patients die from exhaustion and blood-poisoning.

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* This statement by Arlidge, however, is not in accord with the statement by others, most of whom say that the disease is ushered in with a chill and that the temperature rises.

According to the observations of Spear, quoted by Arlidge, the external form of anthrax is the more prevalent on the London docks. Here the hides are handled by stevedores, wharf-laborers, merchants, and carters. Spear calls attention to the difference between this affection in the employees of the London hide trades and the disease in the wool-sorters of Bradford, among the latter the internal forms being the more prevalent. This difference is evidently due to the differences in the manner of exposure, the wool-sorters inhaling the poison with the dust of the air.

The *internal* form may present two varieties. The first of these is the pulmonary, already described. The second is the intestinal. In the latter, which is caused by eating the flesh of diseased animals (and is therefore not necessarily an industrial affection) the symptoms are those of intestinal poisoning. Vomiting, diarrhœa, bloody discharges, cramps, pains in the legs, convulsions, and collapse mark its progress. Constitutional reaction, as marked by chilliness and fever, is sometimes one of its earlier manifestations.

It is needless to say that infection by the anthrax bacillus is not confined to those who work in wools and hides. Shepherds, butchers, farmers, knackers, and veterinarians, and in fact all persons who come in contact with diseased animals, either living or dead, are exposed to the risk. Law (Pepper's "System of Medicine," Vol. I.) says that physicians whose practices include large tanneries see the disease frequently.

The *prevention* of wool-sorters' disease depends largely upon wise regulations. Hime (*op. cit.*) drew up a set of rules which have been adopted in Bradford, England. All suspected wools are thoroughly washed and sorted while damp, or they are disinfected. Specially noxious wool, as that from Van, Persia, Alpaca, and Cashmere, are opened and sorted in a room apart and over a ventilating fan. Experienced workmen are specially selected to do this work. Ventilating fans and shafts are provided, and the dust is collected and burnt. All refuse, and the bags containing the materials, are disinfected before being sold. No person having an open wound is permitted to work. Provision is made for storing the sorter's clothing and food while he is at work. Ventilation, cleaning, and disinfecting the sorting-room are carefully provided for. Requisites for disinfecting and treating scratches and wounds are kept ready at hand. Finally facilities for washing are provided.

While great good, according to Hime, has been attained by these regulations, some of which are admirable, one cannot but deplore the crudeness and absence of scientific methods in some others of them. They raise the question how even an "experienced" wool-sorter is able to recognize the presence of anthrax in wool! This is certainly a scientific problem that cannot be solved by even the most experienced wool-sorter. Possibly these rules are the best practicable up to date. Washing in suds will not disinfect wool or hair, although sorting the wool and hair when damp may tend to prevent the dissemination of the spores. It must be remembered that it is by the spores that the infection probably takes place most frequently. These spores are minute spherical bodies, each one of which hatches out a bacillus, which becomes the starting-point for a vast new growth by the process of segmentation. These minute eggs are formed in the bodies of the parent bacilli, and are exceedingly resistant to poisonous and corrosive agents and even for a while to heat. They may pass unharmed through the intestinal tract; according to Koch, in fact, they can penetrate the intestinal mucous membrane, and thus give rise to the intestinal and constitutional forms of the infection already noted. They are probably taken up by animals in the pastures. When dried and floating in the dust of the air it would seem that efficient ventilation is the surest means for disposing of them.

The *treatment* for malignant pustule is surgical. For the constitutional forms of the infection a sustaining and stimulating treatment alone will avail.

EFFECTS OF TOBACCO.

Much difference of opinion exists on the subject of the evil effects of the manufacture of tobacco. Writers *pro* and *con* have expressed themselves with the greatest positiveness. In considering their ofttimes contradictory statements one cannot but be impressed with the possibility that some of them were looking at and describing very different things from those that others of them saw and described. These differences were probably due to variations in races, countries, and above all in hygienic and trade conditions. There is no reason to believe that tobacco-workers all the world over live and work under identical conditions. Into the question of their health and the influence of their occupation enters also the consideration of many other questions of personal and social hygiene. This is in accord with the fact, already referred to frequently in this paper, that occupation is but one factor, and sometimes only a minor one, in the causation of disease in artisans.

Poisson (*Annales d'Hygiène*, VI., 3 S., 1881, 385) has reviewed this vexed question with care, and his conclusions, based upon statistical study, shall be my guide here.

Tobacco yields a most virulent alkaloid, nicotine. It is not certain by any means, however, what proportion of this, if any, exists in the atmosphere of tobacco manufactories. The vapors that emanate from tobacco when drying or when in fermentation have a strong odor of tobacco, but according to Poisson an analysis of the atmosphere charged with these vapors is still a desideratum. It remains uncertain whether nicotine exists in these vapors, although ammonia and carbonic acid are found in them. It has even been denied by Wohl, and by Eulenburg, that tobacco-smoke contains nicotine; they claim that the evil effects are caused by succinic, valeric, and other acids and such bases as conidine, picotine, etc.

The manner of absorption of the tobacco poison in the manufacture may be, presumably, by inhalation of dust and vapor, and by direct contact and absorption through the skin. The dust and vapor of the workshops, as already said, are charged with the peculiar odor.

Absorption by the skin is certainly possible. The physiological effects of tobacco can readily be obtained by the application of tobacco juice or of a strong decoction of the weed to the skin. I have seen a small dog made very ill by a tobacco-wash, used for killing fleas. It seems that workmen and workwomen, however, come in contact with tobacco only with the skin of the fingers and hands, and the absorbing power of the skin in these places is slight. There is no satisfactory evidence that workpeople suffer in this way. Moreover, many of the workpeople use tobacco to excess, especially in the form of chewing or snuff, and the amount that they absorb thus must be vastly in excess of what they could take in either by inhalation of vapors and dust or by contact with the hands.

Ramazzini was apparently the first to ascribe bad effects to the manufacture of tobacco; he even described some of its sternutatory effects upon horses. He saw this manufacture, however, when it was at its crudest and worst, and when bad hygienic surroundings were responsible for some of the effects. Parent-Duchatalet and Simeon denied any evil effects to the manufacture. Some even claimed that tobacco was a preventive of dysentery and miliary fever. Melier, quoted by Poisson, ascribed to this industry a most happy influence upon the progress of phthisis. Ruef even advised that phthisical patients be engaged in this manufacture in order to enjoy this advantage. Hurtaux, however, from large experience denied that there is any such beneficial effect. These instances are illustrations of the conflicting opinions on this subject. A large number of other writers have engaged in the fruitless controversy.

Then, again, the subject of the influence of this industry upon menstruation, pregnancy, abortion, labor, and lactation has been discussed without much profit. Lefail tried to prove that menstruation is very irregular among the women engaged in this work. Bordier claimed that the evil effects upon maternity were marked, and Delaunay seemingly established, apparently by an inquiry among the midwives, that miscarriages were very common among women who worked in tobacco. It was also claimed by some of these and other observers that the mortality of the new-born children of these women was unusually high.

Poisson in his paper, after examining these opinions, gives the results of his own careful statistical study based upon his observations among the tobacco-workers of Nantes. His conclusions are that the manufacture of tobacco has no influence upon menstruation, abortion, post-partum hemorrhage, or the diseases and mortality of infants; that the bad effects experienced by green workmen, as vertigo, nausea, vomiting, and diarrhœa, which are identical with the sufferings of a neophyte over his first cigar, are of short duration and are not serious; that the cure of phthisis, rheumatism, intermittent fever, and itch is a delusion; that in his experience at Nantes the workpeople seem to be rather unusually subject to gastro-intestinal disorders, as diarrhœa, flatulent dyspepsia, and gastralgia; and, finally, that these workpeople are exempt from chilblains on their hands, which fact he ascribed to their constantly exercising their fingers in their vocation.

According to Tracy (*op. cit.*) the manufacture of snuff is irritating to the mucus membrane of the respiratory passages, and a catarrhal process results, which is characterized by a brown sputum; also a conjunctivitis occurs; but these are seen only in beginners. He notes the case of a man who had worked for thirty years packing snuff in an atmosphere so charged with this substance that the uninitiated could remain in it for but a few seconds. This observer, with Emerson ("Report of the Board of Health of New York," 1874–75), investigated the fecundity of cigar-makers in New York City, and were surprised at the smallness of the families; but these facts may have been due to numerous other causes.

The ill-health, as chloro-anæmia, dyspepsia, asthenia, etc., of many young women engaged in the tobacco industries is probably due to bad food and bad hygiene. From the evidence at hand it does not appear that the manufacture of tobacco is injurious to health.

The Effects of Injurious Environment

EFFECTS OF HEAT.

Excessive heat is encountered in a large number of occupations. It may be the natural heat of the sun or artificial heat in factories, furnaces, etc. There is practically no distinction to be made between the effects of these two varieties of heat; heat exhaustion and thermic fever may arise as well from one as from the other.

Among those who are exposed to the direct heat of the sun, may be mentioned agricultural and all out-door laborers during the summer season. The laborers who suffer most are probably these who are exposed on the streets of our large cities where the air is close and often vitiated. Soldiers on the march in tropical countries frequently suffer.* Frightful ravages have been reported among English soldiers in India. Parker relates the occurrence of such a disaster to a regiment in China, the men of which were heavily clothed and accoutred. While charging up a steep hill many were overcome and fifteen of them fell and died almost instantly. During the Sepoy rebellion in India an English regiment suffered severely on a prolonged march (Barclay).

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* Sennacherib's army is believed by some to have been destroyed by heat ("erethismus tropicus"). See Isaiah xxxvii. 36.

Among workpeople who are exposed to artificial heat may be mentioned especially those working in iron and steel works, in glass manufactories, in sugar refineries, and in weaving sheds. Cotton weavers, as already said, are exposed to a moist heat, which is debilitating, and also to dust. Stokers on large steam-vessels are subjected to intense heat and to coaldust, often in horribly ill-ventilated and dark stoke-holes.

A celebrated case of heat-stroke was that known as the "Bogota Case" (*Glasgow Medical Journal*, 1859–60, VII., 314). A stoker on the steamship *Bogota*, from Rio Janeiro for England, refused to work because of alleged sickness. He was pronounced sound by the surgeon, and still refusing to work was ordered to be confined in the stoke-hole, where he was tied to a ladder three feet from the hottest furnace. One-half hour later his head was seen to droop; he was released and carried to the deck, where he soon expired. The temperature in the stoke-hole in warm latitudes was shown to be sometimes near 150°. The engineer who was responsible for this barbarity was tried, convicted, and sentenced to fifteen years' penal servitude. For the defense it was shown that men worked in the stoke-hole sometimes for so long as two hours without harm; and it was testified that confinement in that place was a common punishment at sea.

The ability of men to endure for a short time exceedingly high temperature has been shown in some alleged instances, which, if true, are indeed remarkble. Fordyce remained for a short time in a temperature of 212° F. without harm. It is asserted that a baker's daughter was once shut up in an oven at 274° F. for twelve minutes. Chantrey, the sculptor, and his workmen sometimes entered an oven that was used for drying moulds in which it is said the temperature was as high as 340°. Some of these instances seem incredible. If they are true they simply demonstrate that the human body can resist for a short time a perfectly dry heat which if long continued would kill. The limit to this toleration is perhaps not strictly fixed. These cases do not prove, however, that a much lower temperature, long continued, in a moist atmosphere, and acting conjointly with hard bodily toil, may not be in a high degree fatal.

This fatality is shown in some of the deep silver mines of Nevada. Mr. J. A. Church (*Scientific American*, November 1st, 1879), in an interesting paper on the Comstock mines, describes the effects of heat. These mines are of great depth, so much as 1,700 and 2,000 feet deep. The hot mine water has a temperature of 158° F., and the hot steamy air one of 128° F. The miners suffer with a confusional delirium, coming on quite suddenly, and leaving, after apparent recovery, a marked loss of memory. A remarkable feature is the tendency of men to faint on going up in the "cage" when they first reach the cool air, about 150 feet from the surface. So common is this that the men are never allowed to go up alone. This reminds one somewhat of the effects observed upon workmen on coming out of a caisson. Long habitude to heat in the mines is no safeguard. Insanity and death are the grave results not infrequently of work in the mines. In one instance a man suddenly became confused and aberrant in mind; he was taken to a "cooling place," *i.e.*, where cool air is forced into the mine; not improving, he was lashed to the cage and taken to the surface, where he fainted instantly and died in a few minutes. It is said that in this case the temperature in which the man had worked was only 90°; but it must be remembered that the air in these mines is moist and contains steam. Sudden cooling on coming to the surface, and drinking ice-water appear to be dangerous, although the miners run these risks constantly. In another instance a new hand went to work one day, worked too hard, against advice, and died the next morning. It seems deplorable, as Mr. Church observes, that no more thorough scientific study has been made of these cases in the Comstock mines. They seem to have been cases of heat stroke, with grave nervous sequelæ, especially psychical changes. It would be interesting to know whether in any cases the cord or peripheral nervous system is involved. The miners themselves, it is alleged (*Medical and Surgical Reporter*, XXXIX., p. 153), sometimes treat these cases successfully with active friction.

Andrew (*British Medical Journal*, May 5th, 1877, p. 548) reports the case of a child who had almost universal paralysis after exposure to great heat in the railroad cars; and Heisler (*Universal Medical Magazine*, 1892) reports a case of heat-stroke followed by multiple neuritis.

In sugar refineries the chief causes for ill health are moist heat, the gases of fermentation, and the contact of the juice and sugared fluids with the skin, especially the hands. The sudden chilling of the body, after the relaxing effects of labor in hot, moist air, induces catarrh of the stomach and bronchi (Kuntz). Deleterious gases are ammonia, carbon monoxide and dioxide, and carburetted and sulphuretted hydrogen. Asphyxia from some of these gases has been reported. Eruptions on the skin—eczema, pustules, boils—are frequently seen. Nivet has described an "impetigo glycosique," which is seen in workmen in these refineries.

The *symptoms* caused by exposure to heat may be divided into those simply of exhaustion and those of true thermic fever (Wood). The former are syncope, coldness of the surface, reduced temperature, and feeble action of the heart. Such cases usually recover. This train of symptoms, however, may be much more grave than Wood and some others, who follow him, have apparently apprehended. This can be seen in the cases of the Nevada miners, in whom a fainting spell on coming to the surface is often the forerunner of grave symptoms and even death. According to Broughham (*Indian Annals of Medical Science*, Calcutta, 1857, VI., 525), whose observations were made in India, the symptoms of true heat-stroke are twitching of the facial muscles, increased heat of the surface, insensible pupils, sometimes contracted, sometimes dilated; quick respiration, the air passing through frothy mucus and causing moist râles, and quick feeble pulse. In some cases anæsthesia of the surface is so profound that blisters are not felt. Some cases are sudden in onset and rapidly fatal—a characteristic noted by some military surgeons (Gordon). Brougham's description suggests that in some cases hysterical symptoms may be present. Gordon noted a tendency to over-action of the muscles. He says that the patients have clenched hands, with thumbs turned in; that they froth at the mouth, and have a congested and bloated face. In grave cases the body temperature is very high and unconsciousness is complete.

The treatment is with cold affusion or cold baths. This is especially lauded by Indian surgeons (Buchanan, Gordon). The latter (*Edinburgh Medical Journal*, 1859, V., 985) recommends arteriotomy (of the temporal artery), counter-irritation to the nape of the neck, and the internal use of croton oil. In Philadelphia, in which city many cases of sunstroke occur during our hot summers, it is customary for our large hospitals to have tents erected in their yards in which the patients are treated, main reliance being placed upon the reduction of temperature with cold affusion, baths, and even the ice-pack.

The respiration and heart's action should be watched with care. Strychnine under the skin is a valuable remedy; and where great prostration supervenes alcoholic drinks must be given, if possible.

EFFECTS OF COLD.

Cold is not met with in industrial life to so great an extent as heat, and where it is met with does not as a rule exert its worst effects. Of course, in severe winter weather all laborers engaged in outdoor work experience some of the effects of cold. This general statement is about all that can be made here. These laborers are of various grades and classes, too numerous to be mentioned in detail.

It is not often, in our climate, that they suffer from the worst effects of cold. One class of workmen, however, who furnish occasional examples in Philadelphia of the fatal effects of cold are street car drivers and motormen.*

Cold is often associated with heat in exercising an evil effect; or rather it alternates with heat or follows it. Thus in some of the occupations mentioned as exposing men to intense heat not a little of the harm results from the imprudence of workmen in passing into a cold air to "cool off." This practice tends to induce catarrh of mucous surfaces, and probably rheumatic affections and, without doubt, peripheral neuritis and possibly even changes in the spinal cord. I once saw a case of locomotor ataxia of rapid onset in a ship carpenter, induced by sudden chilling of the body while overheated on the tropical seas, by drenching the person with bucketfuls of cool water. This man may have been predisposed, possibly by syphilis, although there was no history of it. It is not necessary that the air or water, used for a chilling effect after hard labor in hot air, should be exceedingly cold, although the colder it is the more apt the effect is to follow.†

According to Landois the artificial cooling of warm-blooded animals (rabbits) causes depression, falling of the pulse and blood-pressure, suppression of urine, and congestion of the liver. Later paralysis occurs, and death results in spasms and with signs of asphyxia. In extreme cases artificial respiration raises the temperature, and recovery may follow the application of external warmth.

The local destructive effects of cold, as chilblains and extensive frost-bites, are strictly surgical affections.

EFFECTS OF COMPRESSED AIR.

Compressed air is used in caissons; and these are employed for laying the foundations for heavy masonry, as for piers of bridges, usually in the beds of rivers. Compressed air is sometimes used in submarine work also.

A *caisson* is simply a box-shaped structure, made usually of either wood or iron, and inverted so that it is open at the bottom but airtight at the top and sides. It forms the foundation for the intended pier, which is built, usually of stone, on its surface. As the weight of the masonry increases the caisson sinks until it rests first upon the bed of the river. Since this bed, however, is not a suitable support, the object is to sink the caisson, with its superincumbent weight, through the mud and quicksand of the bed of the river until it rests securely upon the solid rock. The depth of this rock beneath the surface of the river varies, of course, with the location. In order to sink the caisson through the water and wet mud and sand it is necessary to force air into its interior until the compressed air within more than balances the pressure of the water and soft earth without. This pressure varies in exact proportion to the depth to which the structure is sunk. By thus keeping the interior of the caisson filled with compressed air a space is obtained in which men can work; the earth can then be excavated within this air-chamber and from beneath its edges, thus allowing the whole structure to gradually sink until it reaches and rests upon the rock. When, finally, it is firmly set upon the rock its interior is filled with concrete, which becoming hard forms part of the foundation for the pier. As the caisson sinks the masonry is built upon it, layer after layer of stone being added at, or rather a short distance above, the surface of the river. A hollow space or shaft is left in the masonry, which communicates with the caisson at the bottom. This shaft gives entrance and exit to the men going to and coming from their work. In some instances a spiral staircase is erected in it; in others an elevator, worked by steam, is used. At the bottom of this shaft, in most instances, an "air-lock" is constructed, which gives admission to the interior of the caisson. This air-lock has two doors, one communicating with the shaft, the other with the "air-chamber," or interior of the caisson, these doors opening inward. It has valves, one opening from the caisson, the other opening into the outer air. In order to gain admission the outer door is opened, the inner being tightly closed; the workmen, entering the lock, then close the outer door. The valve communicating with the caisson is now opened and the compressed air from within rushes through and gradually equalizes the air of the lock with that of the interior, or "air-chamber" as we may call it. When this equalization is attained the men have only to open the inner door and pass into the caisson or air-chamber, in which they are to work. In coming out the process is reversed; the lock being filled with compressed air from the caisson the men enter it and close the inner door; the compressed air in the lock is then allowed to escape through the valve communicating with the outer air. When the pressure within the lock falls to that of the outer air the men emerge. In some instances the air-lock, instead of being placed at the bottom, is placed at the top of the shaft, a much better plan if the men are obliged to climb up a long flight of stairs, as will be seen.

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* Now that the electric car has superseded the old horse car it remains to be seen whether the motormen in their unnecessarily exposed positions will suffer fatally from cold.

† I do not know of an instance of such effect following the cold affusions for sunstroke; although recently I have seen a case of multiple neuritis following typhoid fever for which the patient had been treated vigorously with cold baths.

The caisson was first used in 1839, by Triger,* a French engineer. In order to reach the rich bed of coal that lies underneath the river Loire—a project which had baffled all others—he conceived the idea of sinking an iron tube through the quicksands, and excluding the water and mud by forcing air to a high pressure into the tube. This he accomplished successfully, and thus introduced an engineering method of incalculable value. In Triger's iron tube, which was seventy feet deep, the air-lock was at the top, thus permitting the workmen to ascend the ladder while still in compressed air.

In 1845 the same principle was applied to the working of a mine at Douchy in France, when, apparently for the first time, a scientific study of the pathological effects was made—by Pol and Watelle.

About 1850 this method of Triger was first employed for sinking piers. Since that time it has been used frequently; but we need note only those instances in which the magnitude of the work gave unusual facilities for the study of the injurious effects of compressed air.

The Caisson Disease.—The first instance of importance was the construction of the railroad bridge across the Rhine at Strasburg. The men were under the medical supervision of François, who has written an interesting paper based on his observations (*Annales d'Hygiène*, 2 S., Tome 14, 1860, 289). Foley ("Du Travail dans l'Air comprimé," 1863) observed the caisson disease at the building of the bridge at Argenteuil.

This country, however, has furnished two examples of bridge-building, by the aid of compressed air, which far exceed in importance anything that has been seen elsewhere. The first of these was the building of the great bridge by Eads across the Mississippi River at St. Louis; the second was the building of the Brooklyn suspension bridge across the East River at New York. Observations were made in the first instance by Jaminet and published in book form; and in the second by Dr. Andrew H. Smith, and embodied in a report to the Board of Directors. These two works supply by far the largest body of data that we possess up to date.†

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* For many facts used in this description I am indebted to Jaminet ("Physical Effects of Compressed Air," etc.) and Andrew H. Smith ("The Effects of High Atmospheric Pressure," etc.). I have not been able to obtain Triger's original paper, which, however, was written from the engineer's, not the physician's, point of view.

† Jaminet's work is marred, unfortunately, by extreme dogmatism and a most illiterate style. It is to be regretted that the author did not take more pains with his clinical and pathological observations, and less with his theories. The work, however, contains an immense amount of crude information.

In the case of the St. Louis bridge four piers were built, one on the western bank and three in the river. Each pier was built on a caisson as already explained; that for the western pier in the river having on top a surface area of 2,600 feet, for the eastern pier 3,600 feet, and for the eastern abutment pier 4,800 feet. The air-chamber in each caisson was only nine feet high, and was divided into three compartments by longitudinal timber partitions intended to support the deck. A shaft in the centre of the pier led, by circular stairs, down to the caisson, which was entered by an air-lock. Before the work was finished an elevator was introduced instead of the stairs, with the result that fewer bad cases happened. The deepest pier was sunk 136 feet below high water, and the pressure attained in the caisson toward the end was 50 pounds to the square inch.*

In the case of the Brooklyn bridge the work was on a still more stupendous scale. The caisson on the New York side had an area on its top of 102x172 feet. It was in effect, as Dr. Smith explains, "a wooden box turned bottom upward." The root of the caisson (bottom of the box) was made of solid timber and was 22 feet thick. The space within the caisson was 9 feet high. On the New York side the caisson was sunk 78 feet, when it reached a solid foundation. At the close of the work the pressure had reached 36 pounds, not as high, it is to be observed, as in the case of the St. Louis bridge. In the beginning a spiral staircase was in use in the shaft, but being found to be exceedingly fatiguing it was replaced by a steam elevator. The air-locks were at the bottom of the shaft, which Dr. Smith considers to have been a serious error, as it required the men to make a fatiguing climb just at the moment when they had emerged from the compressed air and accordingly could least afford to do it. Precautions were taken both at St. Louis and New York to rid the air of the carbonic acid which resulted from the respiration of the men and the combustion of the lights, although these precautions were not taken until after some time. In the New York works serious difficulty was experienced from the inhalation of unconsumed carbon from the lights, the men having cough and black expectoration, which continued in some instances for six months.

It is generally stated by all observers that the "caisson disease" is caused, not by entering and remaining in the compressed air, but by emerging from it into the outer atmosphere. Certain phenomena are observed in the air-lock. In "locking in," *i.e.*, in entering the caisson, it is observed that the temperature in the air-lock rises very notably. At St. Louis it rose sometimes so high as 90°. On the contrary, in coming out the temperature in the air-lock falls rapidly, and this constitutes a serious matter, because, especially in cold weather, the men are thus brought quickly into a lowered temperature at a time when, fatigued with hard work and drenched with sweat in an artificially compressed air, they are relaxed by removal of the pressure.

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* This, as I understand Jaminet, was that much above the normal pressure of the atmosphere, which is 15 pounds. Hence the pressure in the caisson exceeded *four* atmospheres, *i.e.*, 50 pounds *plus* 15 pounds.

At the St. Louis bridge as the pressure increased it became necessary to reduce the number of hours for labor, so that the men worked in shifts for two hours three times a day, having an interval of two hours between periods of work. Later, indeed, toward the very end, the men were allowed to work for only forty-five minutes at a time. At the Brooklyn bridge the men worked at first in two shifts per day of four hours each, separated by an interval of four hours' rest; but later as the depth increased the time was reduced one-half.

Compressed air is also encountered in diving bells. These antedate the use of caissons, but, as a rule, the pressure is not so great as in some of the latter. They are used for laying masonry beneath the water, and are so constructed as to be supplied with a current of fresh air. A diving bell has been made so large as 20 feet square. The men work in shifts for from three to six hours.

Diving suits are now very commonly used to enable men to work at considerable depths. They are used especially for raising treasures from sunken ships. Sponge, pearl, and coral fisheries are now conducted with the aid of these dresses. At 33 feet the pressure is about twice that of the atmosphere. According to Siebe the greatest depth to which a diver, in diver's dress, has descended is 201 feet, which imposed a pressure of 87 pounds on the square inch; but this is an exceptional instance, and this depth is far greater than that which they can endure with safety. It is necessary to regulate the rate of descent so that the pressure may be gradual. About two feet a second has been established as a rule.*

The *symptoms* of the caisson disease are confined almost exclusively to the nervous system. The men, on issuing from the caisson, are usually much fatigued, and, according to Jaminet, have a sallow complexion. When, as was the case at the St. Louis bridge, they are obliged to climb up a long and steep flight of stairs to gain the surface, they are still more exhausted; the pulse is often rapid; and sweating, which was profuse in the air-chamber, continues.

Sensory symptoms are usually the first noted. These may not come on, however, for some minutes, or even some hours, after emerging into the outer air. Among Jaminet's patients a peculiar, severe epigastric pain was felt; this was sometimes, but not often, accompanied with vomiting. Pains in the back and lumbar region, and along the spine, are also experienced. Later, as a rule, severe pains are felt in the legs, beginning usually in or about the knees. In some cases, however, these pains in the leg precede those in the trunk; or the latter may even be absent. All these pains, in whatever region, are peculiarly distressing. Smith describes them as of a tearing character and almost intolerable. They persist even in those cases in which anæsthesia of the part sets in ("anæsthesia dolorosa"). This loss of sensation usually shows itself promptly in severe cases and advances with involvement of the motor system.

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* For further details see the article on "Diving" by Stevenson, in the "Encyclopedia Britannica."

Paralysis of motor nerves is a common symptom. It comes on in a short time (in a few hours or even less) after the initial symptoms are observed; in fact in some cases the motor paralysis is an initial symptom itself. It usually involves the legs, constituting a paraplegia, but sometimes one or both arms are affected. In neither Jaminet's nor Smith's paper (*op. cit.*) is the exact character of the paralysis noted with reference to the electrical reactions and the trophic state of the muscles. Jaminet does not refer to the state of the reflexes in his cases. The bladder and rectum are commonly paralyzed. The catheter is needed for some of these patients; and in fact it is very evident that in some of Jaminet's patients serious infection of the bladder took place from too frequent and careless catheterization and from the practice of letting the patients catheterize themselves. Some of them had discharge of pus from the bladder and fœtid and ammoniacal urine. In the worst paraplegic cases bedsores form.

Although it is claimed by some that the viscera are engorged with blood there are few symptoms indicating serious lesions in them. Jaminet found increased specific gravity of the urine in most workmen under his care, but this he attributed to the presence of urea, and it was a proof to his mind of increased waste or metabolism of tissue.

Brain symptoms are seen in a few cases. Headache is experienced by some in passing through the lock. In some instances vertigo is experienced. In grave cases unconsciousness supervenes early, and in all fatal cases comes on before death. In one fatal case the man lapsed into unconsciousness soon after coming out of the caisson and died in a few hours. According to Smith death occurs only in cases that are severe from the first and show cerebral and spinal symptoms. The duration of mild cases is from a few hours to six or eight days. It seems that the rule is for recovery, when it occurs, to be complete; although from the lesions described by recent skilled observers it is possible that cases might occur of persistent paraplegia.

Irritation of the bronchial mucous membrane, with consequent cough and expectoration, has been noted in men working in some caissons. This irritation was evidently due to the unconsumed carbon in the air from the lights, and was not an effect of the compressed air. In the future it is probable that the electric light will be used and thus obviate this evil. Smith reports a rapidly fatal case of pulmonary congestion occurring after the patient had come out of the caisson.

Jaminet found for the men under his care a rather high average of the specific gravity of the urine. This ranged as a rule above 1.024, reaching in many cases so high as 1.028 and 1.030. This was attributed by this observer to increased urea. No albumin was present in any of the specimens tabulated (*op. cit.*, p. 133). It seems rather remarkable that renal changes were not more marked in these cases, because in one of them Jaminet found post mortem a clot in one of the kidneys.

Perforation of the ear drum and catarrh of the middle ear have been reported. It is probable that the latter is really a primary disorder and that the perforation is its indirect consequence, *i.e.*, a patient having middle-ear disease with obstruction of the Eustachian tube is unable to equalize the air in the tympanic cavity, and hence perforation occurs. Pain in the ears is not an uncommon symptom in passing through the lock.

The pulse during an attack is usually rapid, but slowing has been observed in some cases.

François described symptoms which he attributed to congestion of the liver and spleen. These observations do not seem to have been confirmed by more recent observers. He also observed an intermittent fever, which, however, was evidently not due to compressed air but probably to infection by the malarial organism in the locality in which the work was done.

The exact *pathology* of the caisson disease is somewhat obscure, and, according to Smith, may not be the same in all cases. The subject has been somewhat mystified by a large amount of theory and a minimum amount of exact observation.

François believed that the cause was an excess of oxygen in the tissues. This was the first statement of the so-called *gaseous* theory. It was much elaborated afterward by Paul Bert, who experimented on animals under very high pressure—six and seven atmospheres, which is much higher than men have been subjected to in caissons. He taught that the gas in the tissues was not oxygen but nitrogen, and claimed that bubbles of it could be demonstrated in the blood. This theory has been supported by Catsaris, whose observations were made on the divers for sponges near Greece, who use a diving bell. He also experimented on animals. On the merits of this and other theories I cannot enter into a discussion here. It is enough to say that no one theory has been accepted by all.

Jaminet believed that the symptoms are due to excessive waste of tissue in the compressed air, and the storing of the products of this waste and their imperfect elimination after removal of the men into the outer air. He pointed to the high specific gravity of the urine as the principal proof of this alleged fact. His theory is not expressed clearly and to me does not seem sufficiently established. I have thought, however, in reading the description of his and other cases, that the symptoms do suggest the possibility in some cases of a systemic poisoning by some foreign substance in the blood, and I have thought that carbonic acid, combined with the effects of cold and dampness and congestion of the central nervous system, might account for some cases. We know that hard labor in cold, wet situations, under circumstances in which compressed air is not a factor, excites peripheral neuritis and possibly even myelitis, especially in persons predisposed by strong drink.

Smith appears to believe that a lowered vascular tone, caused by prolonged congestion, is the cause of the symptoms. If mere congestion, however, is the cause it is difficult to understand why it is that the symptoms set in only *after* the patient has come out of the compressed air. It seems plausible to argue that the blood is driven from the surface in upon the internal organs, but the congestion of these must be greatest while the men are in the caisson. Smith meets this objection by the supposition that not the congestion itself but the weakness of the vessels due to it causes a blood-stasis when the external compression is removed and the blood rushes from within outward.

Moxon and Gowers advocated a theory of revulsive anæmia, according to which when the blood rebounds from the cord that organ is left paralyzed by insufficient blood supply, favored by the fact that the vessels to the cord are long and of rather small calibre. But unfortunately for this theory the cord is always found post mortem to be congested with blood.

Van Rensselaer ("The Pathology of the Caisson Disease," 1891) is inclined to accept Smith's theory of a congestion with subsequent blood-stasis. According to this theory, as already explained, block-ing up of the circulation occurs, possibly by small thrombi. Hence a destructive necrotic process (not a true inflammation, as Van Rensselaer suggests) takes place. This author's paper is the most scientific study of the subject which we possess, and being founded on a carefully conducted autopsy with microscopical examination shall be my guide here in a statement of the morbid anatomy of the disease.

Morbid Anatomy.—It is remarkable that previous to the report of Van Rensselaer's case only two spinal cords from cases of this disease had been studied microscopically. Of the twelve fatal cases that occurred at the St. Louis bridge not one was submitted to this examination; while the same must be said of the cases at the Brooklyn bridge, although these were not so numerous. The two cases referred to are one by Leyden and one by Schultze. These cases are abstracted by Van Rensselaer and compared with his own. In his own case the spinal meninges were normal and there was no extravasation of blood. The amount of spinal fluid was normal. After hardening, many parts of the cord were found on section to have undergone pathological changes. Ascending degeneration was found in the columns of Goll and in the direct cerebellar tracts, and descending degeneration in the pyramidal tracts, respectively above and below the dorsal region. A disseminated myelitis * was found in the dorsal region, worst in the mid-dorsal area for about two inches. Scattered through the cord from the level of the twelfth dorsal vertebra upward small yellow spots were found, all lying in the posterior columns and in an antero-posterior position. The centres of some of these spots were necrosed and crumbly. At the region of the greatest change the spots coalesced and occupied the whole of the posterior columns. In this region also the lateral columns were degenerated more extensively than merely in the pyramidal tracts. A few small isolated areas of necrosis were seen also in the anterior columns. The gray matter was normal throughout. No hemorrhage was found. Under the microscope the gray matter was found normal. The cells in the anterior horns were perfectly defined and their processes were clear and distinct, as were also their nuclei. In the yellow spots in the white matter, above described, no axis-cylinders could be found, but there was extensive destruction of tissue. The blood-vessels were enlarged, their adventitia thickened and studded with cells. No cells of Deiters could be found. The neuroglia was increased in thickness. In the parts less involved the nerve-fibres were enlarged and swollen; in some the axis-cylinders were very small, in others they had disappeared. There was no trace of hemorrhage nor any infiltration of cells. The findings in Leyden's and Schultze's cases were practically the same as in Van Rensselaer's.*

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* Van Rensselaer, not unnaturally following the older terminology, calls this process a myelitis. It may be doubted, however, whether it is a true inflammation, *i.e.*, the result of infection. It is rather a necrotic process, as we should expect if the theory of causation advocated by this author is correct.

From the reports of these three cases it seems clear that the lesion in the caisson disease is a destructive or necrotic one. This is most probably due to a deprivation of blood, and must be accounted for on some mechanical principle. What the correct principle is may be still a matter of some speculation. No mention is made of the appearance of thrombi in the vessels, but the vessels are described as thickened and their sheaths rich with cells. It is expressly stated by these observers that no infiltration of cells, and no Deiter's cells, were observed, although the neuroglia was thickened. The destructive process, however caused, when once set agoing sufficiently, has apparently no adequate resistance offered to it, and repair, after a certain point is reached, is apparently impossible. The process seems to be strictly analogous in its results with that which occurs in cases, which are not unfrequently seen in hospitals, of acute softening of the cord in the dorsal region, the exact causation of which is obscure. These cases also have in the past been called instances of myelitis, but they are probably not truly inflammatory. In the caisson disease the milder cases, in which probably the change does not proceed beyond a vascular engorgement or temporary obstruction, tend to recover.

Much can be done for the *prevention* of the caisson disease. Symptoms do not begin, as a rule, to show themselves until a pressure of twenty pounds is attained. The deeper the work goes, *i.e.*, the higher the pressure rises, the more varied and the more urgent the symptoms show themselves. Only strong, rather young men—under forty-five years, and in good physical condition—should be employed. Most observers agree that indulgence in alcoholic drink unfits a man for the work; the use of it between shifts predisposes to an attack. Jaminet is especially emphatic on this point, and he seems to have endeavored, not always with success, to control the habits of the men in this and other injurious indulgences. Full-blooded and fleshy men, according to Smith, are more prone to an attack than are spare men. Laborers with pulmonary or cardiac disease should not be employed; although one of the French observers reports the case of a workman with a large cavity in one lung and hectic symptoms who improved markedly during long-continued work in a caisson.

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* Van Rensselaer's paper furnishes an extensive bibliography, which can be consulted with profit by any one who desires to enter into an exhaustive study of the history and phenomena of the caisson disease.

The hours for labor should be carefully regulated. At the St. Louis bridge the men worked in shifts of two hours each three times a day with two intervals of two hours for rest. Later it was found necessary to reduce this time, but the pressure (55 pounds) attained at that bridge was greater than is usual.

It is highly important that men should lie down and rest for an hour or more after hard toil under very high pressure. This should be made imperative and accommodations should be provided for them. They should be supplied also with some nutrient. Jaminet gave strong beef tea. They should never go to work with an empty stomach. Smith advises a meat diet and the free use of warm coffee.

Great care should be observed in "locking out," *i.e.*, in coming out of the caisson. The expansion of the air in the lock during this process is accompanied with a marked fall in temperature. This acts upon the men when they are fatigued and sweating profusely, and are feeling the relaxing effects of diminishing pressure. Jaminet made a rule that the time consumed for coming out should be one minute for every six pounds of pressure. Thus if the pressure in the caisson was 42 pounds the time consumed in the air-lock would be seven minutes. Although this was a purely arbitrary standard, still if it serves a good purpose it is worthy of being followed. During this fall of the temperature it is highly important that the men should be warmly clad.

It is a great evil for the men to be obliged to climb up long flights of stairs or steep ladders after quitting their work. If a steam elevator is not used the ladder or stairs should be in a shaft continuous with the caisson so that the men could mount it in compressed air. Triger's iron tube was arranged thus, and he speaks of the ease with which the men could climb in compressed air. In the ordinary atmosphere, however, this climb when the men have just come from work in the caisson is exceedingly exhausting. At St. Louis one hundred and seventy steps had to be mounted; but these were afterward replaced by a steam elevator. In a recent engineering work in Philadelphia, the building of the Baltimore and Ohio railroad bridge across the Schuylkill River, the caisson was so arranged that the ladder or stairs was in compressed air.

Provision should be made for carrying off carbonic acid and unconsumed carbon in the air.

The *treatment* for the caisson disease is largely according to the symptoms. For the epigastric pain Jaminet found a stimulating cordial, as Jamaica rum, very serviceable. The mild cases, with epigastric pain, do not require much more than this; in fact, as they tend to recover they do not require much treatment other than rest in bed, sustaining food, and time.

Smith writes strongly in favor of relief for the severe pain. He argues, plausibly, that in cases that tend to recover nothing is lost by relieving the patient's suffering, and that this may as well be done with a full dose of some anodyne. A hypodermatic injection of morphine may be given in a full enough dose to attain the desired result; if one dose does not accomplish this a second smaller one may be given. He found that large doses—one-quarter to one-half a grain—were required, as the intense pain induced a remarkable tolerance for the drug. Smith was led also to try ergot. He was led to this by theoretical considerations of its alleged power to control capillary circulation, and also by the theory which he had conceived, that vascular relaxation is the cause of the affection. Although not always efficacious he found that ergot was very useful in a considerable number of cases. He noted that severe pain was completely relieved within half an hour after the administration of a drachm of the fluid extract.

The hot bath has been used, but Jaminet saw bad results from it and stopped it. Paralysis came on while the patient was in, or after he had just left, the bath.

Cold to the spine has been tried with apparent good effects. Cups also have been used.

Electricity and friction with liniments are not of much if any benefit.

For the paralysis gentle massage and the use of strychnine under the skin are indicated. In my own observations of various acute, but perhaps more frequently peripheral, palsies I have thought that strychnine in this way is rather more efficacious than by the mouth.

The immediate return of the patient into the compressed air has had strong advocates in Pol, Foley, and Smith. Foley considered this a specific. Smith says that he never knew an exception to the rule that pains disappear almost immediately upon the return of the patient into the compressed air. He advocates the construction of an air-chamber, which could be put in communication with the condensers, and thus supplied with condensed air at the pressure desired. A cot bedstead could be rolled into the apparatus, and the patient could be kept under observation through glass windows. Several hours could be taken, if necessary, for gradually reducing the pressure. The very best results, according to Smith, might be expected from this method in cases of extreme pain, or in cases of paralysis not depending on extravasation of blood.

Bert recommended the inhalation of oxygen on the theory that nitrogen gas in the blood is the cause of the disease, and that this gas diffuses readily into oxygen.

EFFECTS OF RAREFIED AIR.

It is but seldom that rarefied air is met with in occupations. With the exception of aëronauts there are few whose calling brings them within its influence. In some of the mines of our Western States the elevation above sea level is very great. In Utah some mines are between three and four thousand feet above this level, but I do not know of any accurate study that has been made of the subject under these conditions. The principal ores mined are those of silver and lead, and the action of the latter would have to be taken carefully into account in estimating any deleterious effects upon the health of the miners.

The effects of rarefied air are rapid action of the heart, shortness of breath, bleeding from mucous surfaces, depression, asthenia, and in some cases even paralysis.

Boyle (*St. Louis Medical and Surgical Journal*, 1851, p. 454) gave a description of the so-called "mountain fever," which he encountered during an overland journey to California in or about 1850. More than half of his company were attacked. They had been riding on horseback for between twenty and thirty miles a day, and had attained a great altitude (between 7,000 and 8,000 feet) when the first case occurred. Their food had been fat bacon and sea-bread, and, under the influence of the rare atmosphere, they had eaten voraciously. The tenuity of the atmosphere was so great that it had caused a constant feeling of want of air. The physical condition of the party, owing to exposure, fatigue, and bad food, was evidently not good. The attack was usually sudden. The symptoms were a sense of vacuity in the head and stomach, unsteadiness in the legs, loss of appetite, violent fever, hard, full, and rapid pulse, furred tongue, flushed face, constipated bowels, scanty urine, pain in almost every part of the body, especially in the head, neck, and loins, and delirium and restlessness. Boyle attributed the disease to a deficient oxygenation of the blood.

Ewing (*St. Louis Medical and Surgical Journal*, 1855, p. 454) gave a somewhat similar description of this affection as encountered by him in crossing the Rocky Mountains. The disease had a distinctly remittent type, and was not encountered below an altitude of 7,000 feet. He says it occurs in miners; in fact, it seems to occur in persons who are exposed to hard toil at a great elevation.

Ewing calls attention to the fact that the ordinary conditions for miasmatic fevers were not present in the circumstances in which he saw the disease. At that great elevation vegetation was rather scant; decaying organic matter, vegetable or animal, was not seen; the water flowed in pristine purity from the snow-clad tops of the mountains; and wet and moisture in the air were unknown.

Wise (*British Medical Journal*, 1880, II., 805) described his own symptoms at Davos at an elevation of 5,105 feet. He had epigastric pains, slight rise in temperature in the evening, and an irritable heart. He says that not a few persons arriving at Davos suffer with these symptoms.

In later years there has been a tendency evidently to abuse the term "mountain fever" and to apply it indiscriminately to the condition known as "mountain sickness" (see p. 201) and to many febrile states, some of which are observed in cases at no great elevation. This has led to a reaction against the use of the term. Dougan, quoted by Curtin (*New York Medical Journal*, 1887, XLV., p. 29), thinks that many cases called "mountain fever" in recent years are cases of abortive typhoid fever, and intimates that this latter fever may be modified by climate. In camp, army, and mountain fever there is sometimes a mixture, according to him, of the malarial and typhoid poisons. Dr. Hall, of the United States army, also quoted by Curtin, has seen men in the field and mountains take this fever. Curtin, after weighing the evidence, does not believe in a distinct mountain fever.

It is significant, however, that not only in the Rocky Mountains but in the Himalayas and in the Andes a somewhat similar type of fever has been met with. In India it is called by some the "hill malaria," a name which seems to indicate that it is merely one of the ordinary intermittent fevers.

Work, of Pueblo, Col. (*Medical News*, 1893, LXII., 376), says that it has long been a much-vexed question among practitioners in the entire territory influenced by the Rocky Mountain climate whether there is such a disease as the mountain fever. Work does not believe that it is a distinct affection. He thinks that a number of affections go by this name, among which are the various specific contagious febrile diseases, as typhoid and relapsing fevers, as well as local inflammations and catarrhal processes of the respiratory and intestinal tracts. "Mountain fever" was discovered by the pioneer hunters, and since their time it has been ascribed by many observers, both professional and lay, to some occult mountain influence. It has been credited with many deaths, but not one constant characteristic lesion has been found post-mortem. Work thinks that most of the cases are instances of simple continued fever. The most common type, according to him, is marked by a high temperature and by cerebral symptoms, and is due to prolonged and exhausting physical exertion. Laborers exposed to the sun get this type of the fever. Above an elevation of 4,000 feet men find it difficult to do a day's work.

To show how loosely the term has been used in our extreme Western States I may refer to the report of a case by Newton (*Philadelphia Medical Times*, 1882–83, p. 7) of a trooper in the United States army at Fort Stanton, N. Mex. The man died from a febrile affection, and the autopsy revealed the intestinal lesions of typhoid fever, and yet the case is reported as one of "mountain fever."

While the term "mountain fever" has been abused, and while perhaps there is no such affection, if we except the acute condition called "mountain sickness," yet the fact remains that a febrile reaction, due apparently to fatigue, exhaustion, and poor food at high altitudes, is occasionally noted in miners, soldiers, mountain-climbers, and others who are obliged to toil in a rarefied air. The exact pathology of this febrile state is unknown, but probably it is, as Work suggests, simply that of a mild type of continued fever. It may be due to some auto-infection or to some defective process of elimination.

EFFECTS OF CONCUSSION OF THE AIR.

There has long been a prevalent belief that occupations that expose men to violent concussion of the air cause deafness. Those especially who are exposed to such concussion are boiler-makers, workmen on sheet-iron, and gunners.

The injury in boiler-making is caused by the riveting, and is inflicted especially upon the man who is stationed inside of the boiler. He receives the full effect of the terrific din caused by the repeated blows of the hammer on the rivet on the outside of the boiler. In some instances boilers are now riveted with a machine worked by steam. I have seen such a machine in operation in Baldwin's Locomotive Works in this city. It is probable, however, that in the larger boilers, as marine and stationary boilers, the old method of riveting is still employed. The action of the cause is facilitated by the small space in which the riveter works, whereby the concussion of the air is intensified, the sound waves being confined within the narrow dimensions of the boiler.

Workmen in sheet-iron works are exposed to almost as much noise as are boiler-makers. A man at present under my care in the Philadelphia Hospital worked for years on sheet-iron, part of the time for the United States Government, and is now suffering with deafness. He says that he has known of other cases among workmen. So many as thirty to forty men would be working in one shop, and the noise was so great as to cause some of them to go home in the evening with headaches. He says further that the noise in some sheet-iron works is almost as great as in boiler-works.

Gunners are said to lose hearing sometimes from the violent concussion caused by the discharge of large pieces of ordnance.

The pathology of the affection, according to Strawbridge (Pepper's "System of Medicine," Vol. IV., p. 840), is probably a paralysis of the terminal endings of the auditory nerve. Buck, quoted by Tracy (*op. cit.*), says that he has noticed in the majority of cases seen by him a milkiness and thickening of the membrana tympani.

Care should be observed to distinguish true boiler-maker's deafness from ordinary cases of chronic middle-ear disease. It is possible that men working in noisy places may suffer with deafness from the same causes as the rest of mankind.

The only means for prevention is to stuff cotton or wool into the ears. Treatment will not be likely to be of avail so long as the cause continues to act.

Effects of Bad Sanitation and Ventilation.

In the cases of many occupations the bad effects upon health are due to defective general sanitation and especially to defective ventilation. This fact has been illustrated again and again in the course of the present inquiry, so that it would seem scarcely necessary to go over the subject once more in detail. It is desirable, however, to group some general facts under one heading, so that the truths they embody may be easily applicable to the innumerable instances that are constantly occurring in the industrial world; many of which, because they are not conspicuous, have no doubt escaped attention in this paper.

Lakeman ("Health in the Workshop," London, 1884) has shown how careful the English law is to step in and regulate not only child-labor, as has already been shown in this paper, and the hours for labor, but also all the details of sanitary construction, ventilation, and operation, in factories. He is able to say of his own country that "everything is tending to sanitative action, to the rescuing of inhabitants of cities and towns from impurities, and for enabling workers to dwell in healthy homes and to be employed in healthy workshops." The picture which he presents in his admirable book, although perhaps slightly overdrawn and highly colored, is in glaring contrast with the glimpses which we get from former writers of the dark and noisome and reeking factory life of a generation or two ago in England. As the evils of factory life, however, and the diseases of occupations are not confined to any one nation it behooves the American sanitarian and physician to see to it that our work-people do not fall into the same extremes of distress and squalor that formerly characterized so much of the factory population of the older country.

Among the evils enumerated by Lakeman, which it is the object of sanitary science to remedy, are the use of dwelling-houses for manufactories, limited space, vitiated atmosphere, overcrowding, the accumulation of refuse, deficient light and defective water, noxious vapors from ill-contrived water-closets, excessive consumption of gas, overheating and inadequate and ill-timed hours for meals.

It can readily be seen how impracticable it is to enumerate all the great and small industries in the United States which present some or all of these defects. A few instances, however, may be referred to of former existing abuses, especially in England.

The cotton mills once were insanitary hives to the last degree. The evil began, according to Lakeman, when steam was introduced as a motive power and when machinery was put on to quicken speed. Then "degeneracy marked the effects of protracted labor—the primitive race of sturdy cotton hands disappeared before a generation of weak and deformed people." The cause for this degeneracy was continuous labor by day and also by night, on deficient food and an excess of tea, and in an overheated air. According to some reports the "sizing" of cotton with China clay filled the factories with a fine and irritating dust, while the air was kept at a high temperature and in a very humid state in order that the thread, thus sized, could be manipulated properly.* No ventilation was provided. The workmen, in carding and "blowing," remained all day in a "cloudy mist." The moist heat of the spinning sheds was obtained by injecting steam into them.

In the flax mills things were even worse. Heckling flax was, and is, a most dusty and injurious trade. Formerly no means were used for ventilation and carrying off this dust. In the process of wet-flax spinning the conditions, though different, were no better. The clothes of the operatives, mostly young girls, were saturated with the condensed steam and spray from the machines, the floor was flooded, and the temperature was raised to 83° (Lakeman). The remedies for these evils are covers for the troughs containing hot water, aprons in front of the machines, and ventilating fans and hoods.

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* This "sizing," or coating with a preparation of China clay, is a fraudulent device intended to make the goods more marketable. According to Arlidge the more inferior the yarn the more heavily it is sized. The practice was introduced by English manufacturers. Inasmuch as it is prejudicial to health it should be interdicted.

In the pottery trade, besides the evils that have already been described in this paper under the headings of "Effects of Lead" and "Effects of Dust," there was great hardship in the excessive use of child labor and from heat. The heat in some parts of the potteries was so high as from 120° to 148°, and into and from out of these parts the employés had constantly to go and come. The time for work was from 5:30 A.M. until 6 P.M., but extra time to as late as 9 and 10 P.M. was not uncommon. Lakeman estimates that one boy, known as the "mould-runner," who carried the moulded utensil into the "stove" or furnace, would carry in one day an aggregate of 3,840 pounds, and would walk eight miles in the same time—and this in an atmosphere reeking with dust and heated in some parts above 120°. These children in some instances were under ten years of age. Whole families engaged in the work together—the father perhaps working at the wheel while his young children assisted in various ways; the whole group tending to a degenerated physical and moral state because of the frightfully insanitary conditions of their labor. The negro slavery of this country never presented such terrible pictures as are shown by some English writers in their descriptions of the factory life in their own island.

The remedies for the evils referred to are to be found in cleanliness, ventilation, sufficient space and light, regulated hours for labor and for meals, the removal of refuse, sanitary closets, and proper personal and domestic hygiene.

The English law is quite explicit in some instances on the details for cleanliness. The methods and seasons for cleaning are even prescribed, as for instance lime-washing in the cases of various factories and workshops. All details involved cannot be described here; they necessarily vary with circumstances. As in the case of all factory laws, they must be enforced by a properly qualified inspector.

The proper ventilation of buildings, especially mills and factories in which the air is laden with dust, vapors, gases, and other impurities, is a science in itself. It is ably discussed in reference to dwellings by Galton ("On Ventilation, Warming, and Lighting for Domestic Use," London, 1884). Ventilation has for its chief objects the removal of tainted air and the supply of pure air in its stead. Galton divides the subject into three heads: (1) Purity of air, (2) Movement of air, (3) Warming of air.

According to De Saussure carbon dioxide (carbonic acid) is contained in atmospheric air in the proportion of from 3.7 measures to 6.2 measures in 10,000 measures. According to Parkes the average proportion is about 4 volumes to 10,000 volumes.* The proportion varies of course with places and circumstances. If the proportion rises about 7 volumes in 10,000, or .7 in 1,000, or .07 per cent., the air becomes impure, and when the proportion reaches 1 in 1,000 the air is perceptibly unfit for respiration.

The air, especially of dwellings, factories, and towns, contains also a small proportion of ammonia. This may be in large excess in factories.

The other impurities, as dust, vapors, and gases, will vary of course according to the manufacturing process in use.

The amount of air to be supplied per hour will vary according to the number of men and lights employed. According to Lakeman a man expires about sixteen cubic feet of air per hour and about one-thirtieth of this volume is carbonic acid. This would vitiate about 1,000 cubic feet. But double this quanity of fresh air probably ought to be supplied. An ordinary gas-burner that consumes three cubic feet of gas per hour uses up more air than three grown persons.

To dislodge impure air and to supply its place with pure air is the prime object of ventilation. In large factories this is best done usually with a ventilating fan or wheel, driven by steam. The pattern and the location of these fans depend upon the objects to be attained. In dusty occupations a fan is most advantageously placed where it is as near as possible to the site of the process raising the dust. At Wetherill's lead works in Philadelphia I saw one of these fans at work immediately in front of the grinding mill, so that the dust as it arose was at once caught and carried away. The fan must of course be connected with a flue, and if the air is dusty or contains noxious vapors or gases this flue must empty into proper receptacles. In processes raising vapors or gases a hood may be used to collect these and conduct them into a flue, a fan being placed at the entrance to the flue.

Warming the air supplied, while not strictly pertaining to the subject of ventilation, is practically so associated with it that it becomes a necessary part of the scheme. In cold weather the fresh air should not be forced into the building without being warmed. This is done usually by passing it through hot-air chambers. Sometimes the air supplied is filtered by being passed through wire-gauze partitions or screens of cotton wool in order to free it from soot and other impurities with which it is contaminated in large towns and cities.

The amount of cubic space for every individual is an important one. It will depend to some extent upon the ventilation, *i.e.*, upon the quantity of pure and warm air constantly supplied. Large space entails extra expense in building, and also in heating and, to a certain extent, in lighting. About 400 cubic feet is given by Lakeman, but this apportionment requires a constant supply of fresh air. In such dusty occupations as grinding cutlery and in pottery, unless the fans for drawing off the dust are very efficacious, I should think that a larger space than 400 cubic feet per individual employed would be necessary. In other words, the quantity of impurities, as dust and gases, set free by a manufacturing process should constitute a factor in estimating the required cubic space in a factory.

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* Lakeman misquotes Parkes to the effect that the proportion is four volumes to *one thousand* volumes.

The subject of overcrowding is practically identical with that of the apportionment of space.

The accumulation of refuse may become a source of constant nuisances in mills and factories. Some kinds of refuse are of course much worse than others. In the Trenton potteries Warman (*op. cit.*) noted that much dust was raised from the clay that had been thrown about carelessly in the wet state, had become dried, and so being trodden under foot was a constant nuisance. Such needless inconvenience from refuse should be guarded against in all factories.

Deficient light and defective water are sanitary defects that are always without excuse. The former is the result of imperfect construction, the latter of an imperfect source of supply. Both of these defects may usually be guarded against or remedied.

Ill-contrived water-closets should be abolished, if need be, by process of law. The laws of some of our American States, as already noted in the digest of these laws published in this paper, regulate this important matter. They require that these closets be not only sanitary, but that separate accommodation, not contiguous, be furnished for each sex.

The excessive consumption of gas in some small and badly contrived work-shops, especially when these are in dwelling-houses, is a great evil. As already said, one gas-burner consuming three cubic feet of gas per hour (and some of them consume more than this) will vitiate more air than will three adults. This evil is to be remedied by proper ventilation, already discussed.

Over-heating should be discussed in effect in connection with ventilation. The evolution of excessive heat is an accompaniment of many manufacturing processes; in some it is apparently inevitable, as in wet flax spinning from the steam, and in potteries from the stoves or furnaces used for baking. In others, as in cotton spinning, it is for no good object; as, for instance, when it is for the purpose of facilitating the working of poor yarn which has been heavily "sized" for purposes of deception. By proper ventilation a portion of the excessive heat can be drawn off.

The hours for labor and for meals are regulated now by law in many countries. This is so especially with reference to the labor of women and children (see section on "Age and Sex in Occupation").

Nothing special need be said here about the use of dwelling-houses for work-shops, and the conversion of old buildings (dwellings, barns, stables, etc.) into these. The evils involved arise from inappropriate construction and imperfect ventilation, heating, lighting, and closet accommodations; and these should be remedied as already indicated.

Many sanitary evils are exhibited in their most aggravated forms in the modern "sweating" system so much in vogue now in our great cities. It is perhaps difficult to control this system, which is the direct result of close competition and the employment of cheap labor, without to some extent curtailing the liberties of employers and employés; and yet it constitutes a crying evil which the law and intelligence of civilized states should be competent to mitigate in some manner and degree.

Effects of Injurious Exercise.

Under this heading are included a variety of affections with which artisans may be afflicted. The vast majority of these affections are limited practically, although not exclusively, to the neuro-muscular apparatus. They constitute the class of the fatigue neuroses. A few affections, caused by over-use, are located in other tissues or organs, as the familiar clergyman's sore throat; these, too, may have, nevertheless, a neuro-muscular element in them. Many slight, and a few quite conspicuous, deformities are caused by excessive use of particular parts or organs. Most of these, however, are rather surgical in character; consequently I shall not attempt an exhaustive description of them, but shall merely refer, rather briefly, to a few which may serve as examples. Finally, the deformities due to constrained attitudes, which are sometimes seen as results of occupations, and which may be ranked properly with the class just mentioned, will also be noted very briefly. "There is no exercise, though never so healthful and innocent," says Ramazzini, "but what may produce great disorders if it be used with intemperance."

THE FATIGUE NEUROSES.

Under this general designation may be included all those affections which are caused by the excessive use of particular associated muscles in highly specialized co-ordinated movements; which are located in these muscles as a rule only during the continuance of these particular movements; and which are characterized by an impairment of the functional activity of the affected muscles, either as a spasm, a tremor, a paralysis, or as a combination of these states. This comprehensive definition includes, as it is intended to do, a great variety of affections, which differ so far as causation is concerned, but which nevertheless present symptoms that are almost identical.

The objections to this general term lie in the vague meaning that is attached to the word "neurosis." To these objections I am thoroughly alive, and I do not mean, in employing it here, to commit myself to any particular theory of pathology which the term may suggest. It is not probable that any disease is purely *functional* in the sense that there is not an organic change at the basis of it. It certainly does not seem probable that such obstinate affections as writer's and telegrapher's cramps, associated as these are reported to be in some cases with muscular atrophy, should not have distinct organic changes in nerve and muscle-cells as the foundation for them. I use the term "neurosis," therefore, simply as a convenient label for a variety of nervous affections in which the pathology is still rather vague and the morbid anatomy still unknown. For the general class here designated the term is more convenient in spelling and more euphonious than the various Hellenic and Latin words so far compounded.*

The occurrence of these fatigue neuroses has vastly increased in frequency in modern times. This is due entirely to the increase in number of small machines and appliances which demand the almost unceasing exercise of particular associated muscles. The most familiar examples are of course the telegraphers' key and the typewriting machine. In a sense, however, even old familiar instruments, like the pen, are producing these affections in greater abundance than formerly. In the case of the pen, I do not believe that this increase is due so much to the substitution of the steel pen for the quill as to the increased speed and recklessness with which the former is driven in our modern struggle for existence. This, however, raises some important questions in etiology and pathology which will be discussed later.

The Laryngeal Affections of Professional Voice-Users.

Under a somewhat similar heading Wyllie, in his recent work, ("The Disorders of Speech," Edinburgh, 1894), has given a description of the fatigue neurosis of the larynx which occurs in clergymen, singers, and others who use the voice in public. The subject has been investigated more thoroughly in Germany than elsewhere. Wyllie refers especially to the work of Gottstein and of Fränkel. As I have had but little experience with this particular disorder, which usually falls to the care of the laryngologist, I shall rely to some extent upon the writings of these German authors and of Wyllie. I desire to call special attention to the fact, however, that this disorder in the larynx follows closely the type of the fatigue neuroses as observed in the arm, hand, and fingers, and that its pathology is evidently the same. Therefore it is practically the same disease with which I am familiar in the hand, only in a different organ. It also has its affinities, well marked in some cases, with the intention psychoses and with hysterical aphonia, although in the vast majority of cases it is not identical with these.

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* As examples of words coined for the general class and for some special subgroups, I may mention *copo-dyscinesia*, *graphospasmus*, *mogigraphia*, and *anapeiratic paralysis*.

This fatigue neurosis of the larynx is seen especially in public singers; according to Fränkel it is more common in them than in clergymen. It occurs, however, in clergymen with more relative frequency than in other public speakers, but it must not be confused in them with the chronic pharyngitis that constitutes the ordinary "clergyman's sore throat." These two conditions, however, *i.e.*, the laryngeal neurosis and the pharyngeal catarrh, are often associated. The pharyngeal catarrh is the more common, and is much more likely to exist without the laryngeal neurosis than *vice versa.*

Among others who occasionally have disorders of speech due to their occupations are auctioneers, street-hawkers, military officers, and in fact many persons who are obliged to use the larynx in loud and strained vocal exercise.*

Wyllie discusses the principles and mechanism of voice-production. He calls attention to the important fact that public-speaking holds an intermediate place between song and ordinary speech. "It contains less music than song, but more than ordinary speech, since its tones must be fuller, rounder, and louder, if they are meant to travel to a distance." It is in the skill to produce just enough of this musical tone, and not to overdo it so that the voice falls into a "sing-song intonation," that the art of good public *speaking* lies. This indicates what an extreme nicety of adjustment of fine muscular movement, and what a risk of over-use or strain of this fine muscular mechanism, characterize these vocal efforts. It is in these two features, *i.e.*, delicacy and intricacy of fine muscular coördination and the abuse of this mechanism by over-use, that lies the explanation of all the fatigue neuroses. In these respects they are all identical. It is not necessary to explain them, when they occur in the larynx, by a special reference to obscure physiological processes of voice-production, as Wyllie attempts to do. They are caused there, as they are caused in the hand, simply by an abuse of an exceedingly fine muscular apparatus.

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* Ramazzini, who has a long chapter on the diseases of music masters, etc., includes in the list of those who suffer from over-using their voices, "Chaunters, Preachers, Monks, Nuns (who sing perpetually in the churches), Hawkers, Criers, Clerks, and Philosophers that dispute warmly in the schools." He says that all these are subject to ruptures from the strain caused by using their voices, and he refers to a famous opera-singer of his time who suffered with her voice after singing.

According to Fränkel (*Deutsche medicinische Wochenschrift*, Feb., 1887) this laryngeal disorder has three types: (1) the spasmodic, (2) the tremulous, and (3) the paralytic. In this respect it conforms exactly to the rule governing all these neuroses. As is too often the case, however, with rigid classifications, this one does not conform strictly with the facts. The truth is, that these several types are often associated in the same individual, and in some cases they form stages of the disease rather than distinct varieties. This is so in writer's cramp, in which the spasmodic symptoms are apt to appear early while the paralytic phenomena occur late in the disease. I have never seen a pure tremulous variety.

Fränkel's first, or spasmodic, variety is, however, well represented in these laryngeal cases. It is characterized by a sudden spasm of the glottis; this causes a sudden stop in the voice, quite involuntary, during public singing or speaking. This stoppage may be complete or incomplete; in the latter case the individual emits, with a straining effort and a congested face, altered sounds, sometimes feeble in volume, and always abortive. Wyllie attributes this spasm to a closure of the false cords over the true.

The second, or tremulous, variety of this laryngeal neurosis is, according to Fränkel, characterized by the emission of an involuntary *tremolo*, but he himself does not appear to have made many observations of it.

The third, or paralytic, variety is apparently, in Fränkel's opinion, the most important. The patients may be otherwise in perfect health, and have no disorder in the larynx except when they use it for public song or speech. He describes the case of a young lady whose voice in the midst of a song would break down, gradually failing her until she was forced to stop. This collapse was attended apparently with an emotional crisis, for the patient, with tears in her eyes, would protest that she could not go on with her song, although in so protesting she spoke in her perfectly natural voice. This emotional outburst seems to me to be a more important feature of the case than Fränkel estimates it to be. Another case, in a preacher, was marked by the patient's inability to finish the service. These and similar patients complain of a sense of fatigue in the throat.

The conspicuous mental element seen in certain of these cases of paralytic aphonia does not appear to have struck some of the observers. Thus Morell Mackenzie, quoted by Wyllie, reports the case of a fish-hawker who lost his voice suddenly after he had been using it in excess in his vocation. His aphonia lasted for four months and then was promptly cured with electricity. A somewhat similar case occurred in a preacher, who lost his voice suddenly in the midst of a sermon, and who was promptly cured two months later with a few applications of electricity. It would be satisfactory to know whether these patients had any other possible hysterical stigmata; their cases strongly suggest an hysterical aphonia both in the suddenness of the onset and the promptness of the cure. The *absoluteness* of the aphonia is also in favor of an hysterical origin for them.* Wyllie reports a case, also in a young clergyman, in which the patient had distinct psychical manifestations. In the midst of reading the service he would be taken with dyspnœa (and apparently with palpitation of the heart), and with a distinct *globus* ("a sensation as if the tube were inclined to close"); and he had difficulty in pronouncing individual letters, such as *g*, and could not pronounce *o* without aspirating it. This latter trick is suspiciously like one of the intention-psychoses which are marked by inhibitory conceptions.

That these cases are more common in persons with a neurasthenic tendency is very properly pointed out by Wyllie. The term neurasthenia, however, is now made to include so many nervous phenomena that it does not always convey a strictly limited meaning. From the cases alluded to, and from others seen and reported, I should say that the paralytic or aphonic cases in many instances are strongly suggestive of hysteria, although I do not wish to be understood as saying that I believe that *all* these cases are hysterical. A more critical examination is required to determine whether all of them are so or not. I do not quite agree with Fränkel and with Wyllie that these aphonic cases have a strict parallel in the paralytic form of scrivener's palsy. Many of them are of *sudden* onset and of equally sudden disappearance, which is not so with the affection in the arm. This latter is gradual in onset, and usually has a history of a precedent stage of spasmodic action. Were it purely paralytic from the start, and of sudden onset and characterized by an initial emotional crisis, I should be strongly disposed to regard the arm affection, as I regard tentatively the laryngeal affection, as hysterical.*

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* Chiron, it is known, insisted upon anæsthesia of the laryngeal mucous membrane as a pathognomonic sign in hysteria. It should be searched for in all these cases of aphonia; but it does not seem to have been thought of by some of the observers. Anæsthesia of the epiglottis in hysterical aphonia is described admirably by Sawyer (British Medical Journal, Oct. 31st, 1874). "We may pass the index finger over the patient's tongue," he says, "and tickle her epiglottis vigorously for some moments without causing spasm, choking, or any uneasiness whatever." He says that he has demonstrated this condition over and over again. He is quite sure that a blunted sensibility extends far beyond the epiglottis, and involves also the pharynx and palate. He speaks of how well these throats tolerate the laryngeal mirror. A beginner can have no better patient to practise on than a good-natured woman with anæsthesia of the epiglottis and pharynx.

The *treatment* for all varieties of these laryngeal affections is often unsatisfactory. Fränkel's treatment by what he calls massage, *i.e.*, stroking the skin over the larynx with the finger, appears to me to be rather puerile; and if it cures, probably does so by exerting a mild sort of hypnotic suggestive influence. The same may be said of electricity; cases that it cures promptly are in all probability hysterical, and hence are easily susceptible to suggestion. These considerations are of course in favor of the treatment and not against it. Rest from professional work, *i.e.*, absolute rest of the voice from the injurious exercise, is imperative. Attention to the general health is important. I should be strongly in favor of hydrotherapy and of change of scene.† Finally, good mental hygiene, I believe, is most important for these patients. They should be strongly impressed with the belief that they will recover.

Writer's Cramp.

This is the most familiar example of the fatigue neuroses, and may serve as a type for all of them. It is as unfortunately as it has been variously named, because not one of its familiar names accurately designates it. Thus Gallard ("Crampe des Ecrivains," *Progrès Médical*, 1877, 503) has said truly that the spasmodic action of the muscles is not a true cramp because it is non-painful. If we call the affection *scrivener's palsy* we are met at once with the objection that true palsy is present in a comparatively small proportion of the cases. Such terms as *dysgraphia*, *graphospasmus*, and *mogigraphia* are pedantic and unfamiliar. The term which I have adopted, *writer's*

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*I have seen some remarkable instances of hysterical aphonia: they were not caused, however, by abuse of the voice. One of them lasted for more than six years, and then was cured rather promptly, the first sound of the natural voice returning one day while the patient was laughing.

† It is worthy of remark that Galen (quoted by Ramazzini) says that "those who hurt their voice by over-straining it, such as musicians, preachers, and actors, make much use of baths, etc."

cramp, is well known, has the sanction of long use, and if not accurate, as Gallard claims, nevertheless stands for an affection which if not a true cramp is of a cognate species of motor disorders. Moreover, it indicates the type of the affection that is by far the most common.

Writer's cramp may be defined as a neuro-muscular disorder, characterized by disturbed motility in the muscular mechanism used for writing. Its exact pathogeny and its underlying morbid anatomy are not known. It is essentially a fatigue neurosis—a term which has been already defined.

The *causes* of writer's cramp are predisposing and exciting. The former are not easily estimated, and their influence has sometimes been exaggerated. It may be said, as was said in the case of similar laryngeal affections, that the neurotic or neurasthenic temperament is the chief predisposing cause. Yet I believe that it is a mistake to think that in all cases of writer's cramp the patients are neurasthenic. Many of them are not so. It might almost be claimed that any person could develop this affection who was subjected to prolonged abuse of his muscles. It is enough to say that those who naturally have an unstable nervous system are probably more predisposed to it. Poore (Medico-Chirurgical Transactions, LXI., 1878, p. 111) saw several instances in which this disease seemed to be hereditary.

Lewis ("Neural Disorders," etc., Pepper's "System of Medicine," Vol. V.) claims that the manner of holding the pen is important. When the body of the pen-holder is allowed to fall below the head of the first metacarpal bone, the action of the joint is more restricted and hence more fatiguing than when the pen-holder is kept against the body of the first phalanx.

The kind and pattern of the pen undoubtedly have importance in the etiology. A stub-pen is better than a sharp-pointed pen for persons who are obliged to do much writing. The reason for this is in the fact that a stub-pen does not require such fine muscular adjustment to keep it in proper contact with the paper as that which a pointed pen requires. If any one needs to be convinced of this let him compare the two actions by writing a few sentences first with one pen and then with the other. The handwriting of one who uses a stub-pen is usually more compact and more steady than is that of one who writes with a sharp point. This compactness and steadiness represent just that much saving in the wear and tear of the neuro-muscular apparatus. I have no doubt that the reason why an increase in scrivener's palsy was noted after the introduction of the steel pen was because the latter, when first adopted, had a sharp point in marked contrast with the rather blunt and always soft nib of the old-fashioned quill. There can be no other reason, unless it be in the additional fact that commercial and manufacturing schemes increased vastly about that time, and employed a vastly increased number of hand-writing clerks. The lead-pencil, for practically the same reason as that in the case of the stub-pen, is more restful to the hand than a sharp-pointed steel pen.

The excessive use of alcohol and tobacco is undoubtedly a predisposing cause. In an inquiry among telegraph operators, whose cases are strictly analogous to those of scriveners, I was struck with the almost universal testimony that the free use of these two substances increased the evil.

It does not seem to have been satisfactorily determined by statistics that age and sex are determining causes of any of the fatigue neuroses. Of course, more persons under or about thirty years of age, and more men than women, are afflicted, for the very obvious reasons that more persons at these ages and more men are employed in hand-writing than at any other ages or than members of the other sex.

Lewis refers to albuminuria as having been observed by Mitchell in two cases. I should suggest that a sharp lookout be kept for gout in persons advancing toward middle life. As Poore has shown, and as I myself have seen not unfrequently, disorder in the function of writing is sometimes an early symptom of some deeper-seated, even organic, disease.

Finally any fault in hygiene, personal habits, or the state of the health which depletes the general strength may predispose to this fatigue neurosis.

The one exciting cause of writer's cramp is excessive writing. I am not aware that any exact study has ever been made of the actual amount of writing, and the number of hours per day, that have acted in particular cases. It would be interesting to know the resisting power of individual men, and how much of a tax they had put upon the nerves and muscles before these began to give way. It is probable that the factors vary within wide limits, and that contributing causes, as personal habits and the state of the general health, vary also in their activity.

The *symptoms* of writer's cramp are spasm, tremor, incoördination, paralysis, pain, anæsthesia, and vaso-motor disorders. It is customary with some writers to describe three varieties or types of the disease, *i.e.*, the spasmodic, the paralytic, and the tremulous. As said before, when discussing this same neurosis in the larynx, this classification does not conform strictly to the facts. These various symptoms occur in varying degrees in different individuals. It is true that the spasm predominates in some cases and the paresis, probably, in others; but nevertheless it is rare, if not impossible, to find cases in which spasm alone or paresis alone or pain alone characterizes the type. The truth is, at least in my observation, that paresis is more likely to come as a late phenomenon in cases which were more purely spasmodic in their early stages. Again, tremor is rather a rare symptom. I have never seen a case purely of this type. Pain is a varying symptom and does not appear to follow any definite law. In view of these clinical facts it seems to me better to study the symptoms rather than the types of writer's cramp.

The spasmodic action occurs, as a rule, only in the muscles directly concerned in writing. The actions of these muscles have been carefully analyzed by Poore, and their relative degrees of involvement indicated. The order in which they are involved, according to him, is as follows: The interossei, the extensors of the thumb, the flexor brevis pollicis, the abductor pollicis, the flexor longus pollicis, the adductor pollicis, and the opponens pollicis. This list, it will be seen, while comprising all the small muscles immediately concerned in writing, or rather in holding the pen, does not include the muscles of the forearm, of which Poore speaks as having seen them involved in only two cases out of thirty-two. This is too small a proportion, in my belief. The extensor muscles of the fingers and wrist are important muscles in writing, and among the earliest to feel the effects of fatigue, as any one can observe who notes the condition in his own arm after prolonged use of the pen.

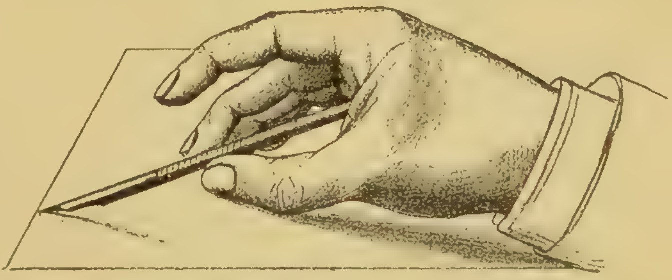

FIG. 4.—Abnormal Pen Prehension. Fibrillary tremor of first right dorsal interosseous. (Poore.)

This spasm may be slight, involving only the small muscles that hold the pen and only during this act, or it may be severe involving many of the forearm as well as the hand muscles (and even the deltoid), and persisting for some time after the initial act has become impossible and the attempt to perform it has been abandoned. The appearance of the hand is quite characteristic; it is held in a vice-like rigidity, the muscles standing out prominently.* This spasm of course interferes effectually with the patient's writing in his usual hand; he sometimes makes a shift to write by seizing the pen in an unusual or abnormal way (see Fig. 4). This spasm is quite involuntary and may resist for some time the patient's attempt to relax it. It is as a rule non-painful. The abnormal methods of holding the pen only serve as a rule for a short time; they lead to grotesque handwriting and sooner or later they excite spasm just as did the normal method. The assertion that the muscles are thrown into spasm by the attempt at writing only, and that they do not become contractured during other fine movements, must be taken with much allowance. Gallard called attention to this fallacy; he instances the act of rolling a cigarette as a difficult one for the patient with writer's cramp to perform.

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* See illustration of telegrapher's cramp in the next section.

In some cases there are widely associated spasmodic movements, as for instance in the foot or in the other hand and arm. Attempts to educate the sound hand and arm usually excite some feeling of discomfort in the affected limb. Thus Solly ("Surgical Experiences," article on "Scrivener's Palsy") says that one of his patients told him that he would prefer not to attempt to write with his left hand, because if he did so he felt an indescribable tingling feeling in the damaged hand, which did not subside on leaving off writing.

Tremor, pure and simple, is rarely seen in true writer's cramp. It is the custom of many authors to make a distinct type of the affection as distinguished by tremor, and yet they almost unanimously say that either it is rare or that they have not seen it. It seems that when it occurs it is seen most frequently in the forefinger. It is possible that some of these cases have not been cases of true writer's cramp, but rather of some form of muscular atrophy or some central nervous disease characterized by tremor or fibrillation. As I shall show later, some organic diseases of the central nervous system have among their earlier symptoms interference with the act of writing, and it cannot be doubted that such cases have been confused sometimes with the disease that we are now considering. It is easy to confuse a jerky incoördinate movement with tremor, and I think it is probable the former that has been meant by some writers.

Incoördination and jerky, choreiform movements are not unfrequently seen in writer's cramp. Gallard speaks of disordered movements, incoördination and ataxia as seen in one of his patients. Duchenne made of these choreiform movements the distinguishing signs of one of his groups. This jerky to-and-fro movement is the natural result of the spastic state of the muscles. As these tend to become "set" or contractured the fingers and hand are pulled about in an awkward and incoördinate manner.

Paralysis is not so common a symptom in writer's cramp as spasm and contracture. It is sometimes associated with spasm, *i.e.*, some muscles may be spasmodically contractured and others may be paretic. It sometimes follows spasm as a sort of terminal stage. This is more common than for it to appear as an early symptom. The purely paretic form of this fatigue neurosis is so rare that it ought to excite careful scrutiny into its genuineness. Some cases reported, coming on rather suddenly after some emotional crisis, are suspiciously like hysterical paralysis. This raises an interesting point in the etiology of hysteria, which cannot be much more than noted here. This has reference to the possibility of hysteria being excited by the same cause that excites the fatigue neuroses, *i.e.*, the excessive use of associated sets of muscles. When the paralyzed arm is the seat of anæsthesia, especially the segmental or gauntlet form, the likeness to hysteria is still more complete, and its identity may even be claimed in some cases. Some authors describe forms of neuritis, with muscular atrophy and paralysis, as among the varieties of writer's cramp. Poore[*] makes a special "neuritic" group. I can only say that cases of true neuritis, with trophic changes, paralysis, and reactions of degeneration, are not cases of what we ordinarily call writer's cramp or fatigue neurosis. To confuse the two does not seem to be excusable. I doubt whether the excessive use of the muscles in writing ever brings on a true neuritis; and hence I hesitate to include any case of neuritis among cases of writer's cramp or scrivener's palsy.

Pain, or a painful sense of fatigue, is present in many cases. This is an early symptom in some patients, and may be associated with a sense of numbness or tingling. Painful spots, especially over nerve-trunks, are observed sometimes; these do not require to be explained by a neuritis, as is done by Lewis and by Poore, but are rather to be referred to the altered nutrition of the nerve-trunks and muscles which obscurely underlies this affection. As a rule the cramps in the muscles are not painful, in this respect differing markedly from ordinary muscular cramps. Exceptions to this rule, however, probably occur. The most purely neuralgic form of this affection which I have seen occurred in a lady, who noted as her first symptom, while doing clerical work, a pain in her right thumb, which extended eventually to her arm and back. After prolonged rest and treatment she resumed work, this time as a teacher, but found that the pain returned and troubled her, especially at night. Later a little weakness of the fingers was noted when they were fatigued with writing. A slight sprain and more and excessive writing caused the pain to become obstinately located in the wrist, making writing and all motion painful. There never was any cramp, tremor, nor incoördination in the affected hand or arm.

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[*] It is very evident, in fact, that Poore's tables contain references to numerous cases that are not instances of this fatigue neurosis. He seems to have taken cognizance of any and every disease in which writing was affected.

Anæsthesia has been noted by some observers in writer's cramp. Gallard saw it following contractures and followed by ataxic movements in one of his cases. Duchenne describes one of his groups as characterized by cutaneous paralysis, by which is evidently meant anæsthesia. Poore ignores the subject entirely in his elaborate tables; he does not seem to have even looked for anæsthesia, although he is careful to note other alterations in sensation, especially pain. Lewis speaks of having seen one case of impaired sensation in the ulnar distribution. Erb, however, is probably correct (Ziemssen's "Cyclopedia," Vol. XI., p. 345) when he says that true anæsthesia can seldom be demonstrated in writer's cramp. I have never seen an instance of it, and considering the extreme likelihood that hysterical symptoms may complicate some of these cases, or be mistaken for them, I should be disposed to regard anæsthesia as an hysterical phenomenon. There is nothing in the pathology of these fatigue neuroses which we know of that could determine a true cutaneous anæsthesia. The action of the cause is almost exclusively to determine a disorder of motion, as spasm, incoördination, paresis, and, in rare cases, trophic changes in the muscles. On the other hand anæsthesia is a not uncommon symptom of hysteria, and may be associated with hysterical paralysis and contracture; hence the necessity for caution not to confound this disease with any one of the fatigue neuroses.

Vaso-motor and trophic disorders are sometimes seen in writer's cramp. A general hyperæmia or turgescence of the limb may accompany the cramp-like action. Flushing of the face and neck is also seen occasionally. A "professional muscular atrophy" has been described, and its existence confirmed by such observers as Eulenburg, Onimus, and Gowers. Smith saw it in blacksmiths, Gowers in a young artist, Betz in saddlers, Gull in a tailor, Friedreich in a dragoon (from holding the reins), Voisin in a morocco-worker, and Scheuppel in a player on the bass-viol.* All these observers apparently thought that the excessive use of associated muscles determined a muscular atrophy. Considerable care, however, is requisite for determining this point. In any particular case of muscular atrophy coming on in an adult it might be possible to see an apparent connection of the disease with occupation, and yet the appearance might be illusive. I am exceedingly sceptical about the occurrence of changes in the finger-nails of a true trophic character in writer's cramp.

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* See Eulenburg's paper on "Progressive Muscular Atrophy" in Ziemssen's "Cyclopedia of the Practice of Medicine," vol. xiv., American edition, 1877, for more details.

Increased excitability to both the faradic and galvanic currents is observed in many cases of true writer's cramp. This seems to be associated especially with the spasmodic disorder. In advanced cases, especially in those in which there is a tendency to paresis, there may be decreased electro-excitability. The reactions of degeneration are not seen in this affection. If they are observed in any suspected case they should suggest at once the possibility of the presence of some other affection, especially a neuritis due to some undiscovered cause.

Mental and moral symptoms are seen in many cases. They are such especially as would be likely to be caused in highly sensitive or neurotic people, as, for instance, anxiety, depression, and hypochondriacal and hysterical phenomena. They are not of great clinical importance as a rule, although in some cases, especially with reference to hysteria, they present problems for diagnosis.

The *diagnosis* of writer's cramp is not difficult. The symptoms as just described, following the action of the cause, are usually unmistakable. Still mistakes do occur, and confusion exists in the minds of not a few writers as to what constitutes writer's cramp. Poore is the most conspicuous example of a writer thus confused. His paper is founded on the observation of seventy-five cases, many of which are evidently not cases of true fatigue neuroses. The diseases most likely to be confused with writer's cramp are those that impair, especially in their early stages, the power of writing. Among these may be mentioned paralytic cases with aphasia and agraphia, general paresis, degenerative cases marked by tremor, fibrillation, or incoördination, as disseminated sclerosis, locomotor ataxia, progressive muscular atrophy, and alcoholic and metallic poisoning; also chorea, and the various forms of neuritis.* It is only necessary to mention these in order to put the average physician on his guard. Hysteria, however, is probably not so easily distinguished from writer's cramp. Some points of differentiation have been given in the description of the symptoms. For other points it will be

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* As an example of organic cord-disease, probably syphilitic, in which the power of writing was abolished early, may be mentioned incidentally the case of a man in my ward at the Philadelphia Hospital. Four years after contracting syphilis he began to have incoördination in the hands, affecting his writing so that he lost his position as a bookkeeper. Later, however, incoördination in the legs and spastic paraplegia developed, and he is now totally unable to walk. The early symptoms in the case, happening in a bookkeeper, might easily have suggested the incoördinate form of scrivener's palsy.

necessary to consider the whole array of hysterical stigmata.* This obviously cannot be done here. The most common of these that simulate writer's cramp are tremor, paralysis, and segmental anæsthesia. The spasmodic action of writer's cramp is not exactly like the contractures of hysteria. The former is excited only by attempts at performing the customary professional act or some act that brings into play the same muscles, while hysterical contractures are not as a rule so excited and, being established, usually persist much longer.

A guarded *prognosis* should be given in every case of writer's cramp. In early cases it is customary to offer a not irrational hope that the disease may be cured. It is exceedingly doubtful, however, whether the disease is ever cured to the extent of permitting the patient to resume the same kind and degree of work as that which precipitated the original attack. Writer's cramp and the allied fatigue neuroses are signs that the extreme limits of endurance have been reached in the particular set of muscles involved, and beyond these limits it will never be possible to go. If work is forced, other muscles will become involved, especially if artificial methods of holding the pen are adopted; and if the other hand is educated it, too, will ultimately become involved. The necessities of most patients compel them to continue at work with what artificial support they are able to obtain; and to supply this support is practically what physicians are asked to do in most cases.

The *pathology* and *morbid anatomy* of writer's cramp are as yet practically unknown. The disease is still included among those affections that are called provisionally the neuroses. This does not deny to them a morbid anatomy; it merely announces that this is unknown. As I have already said, it does not appear to me probable that a true neuritis is the cause of these symptoms. In cases in which there is a true neuritis this has probably been caused by some genuine *materies morbi*, and the interference with writing which it entails has been strictly secondary. Many theories have been elaborated to explain the disease and to locate it correctly. Some writers have held that it is a purely muscular affection; others that its seat is in the peripheral nervous system; others again that it is in the spinal cord, and others, finally, that it is in the coördinating centres in the brain. Lewis (*op. cit.*) has reviewed these various theories. They seem, in most cases, to be the results of barren speculation. Autopsies are almost unknown in true writer's cramp, and probably, even if they were more frequent, would fail to throw light on this obscure question. It seems to me probable that the affection is primarily in the peripheral neuro-muscular apparatus, and that later it is reflected through the centres in the spinal cord, as when it tends to spread from one limb to another.

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* I have described these stigmata in detail in the paper on "Hysteria" in Dercum's "System of Nervous Diseases," Philadelphia, just now in press (1895).

The *treatment* for writer's cramp may be divided into prophylactic and curative.

The prophylaxis of writer's cramp has really a very limited field, for the obvious reason that physicians are usually not consulted until the disease has declared itself. Hence the minute instructions given by some authorities about the exact manner of best holding the pen, and about the angle at which the paper is to be surveyed, do not seem to me to be practically of very great importance. These directions, moreover, are not always practicable, and even if they were, would probably not be so efficacious as those who advise them appear to think. A man's manner of holding his pen is about as natural to him as is his voice, and about as difficult to be altered by mere advice. This is so especially in the cases of men who have used the pen habitually for years. It does not appear proven either that by merely suggesting some particular manner of holding a pen, which seems good to some particular physician, this danger will be averted. I have more confidence in the prophylactic power of a good stub-pen than in aught else. I think that this matter is so important that I would advise all persons who are obliged to do constant writing to accustom themselves to the use of such pens. Their prophylactic virtue lies in the fact that they do not require such a fine and sustained muscular adjustment to keep the point of the pen upon the paper and to guide it along the surface as does a sharp-pointed pen. The character of the handwriting done with one of these pens, in its regularity and compactness, as said already, is an ample proof of the truth of this statement. Next in importance is a large-sized pen-holder. The gigantic cork affairs, sometimes seen, have much to be said in their favor. They act on the same principle as the steel pen; they diminish the necessity for and the strain of fine muscular adjustment. The victim of writer's cramp intuitively aims to relieve this strain by holding his pen in unnatural and grotesque positions. The large pen-holder, if its use is adopted early, tends to accomplish for him what he thus seeks, often in vain, to accomplish later, *i.e.*, relief from strain. I think that a soft rubber coat on the pen-holder might be a preventive if used early, by diminishing the necessity for a firm grip upon the holder. Lewis recommends holding the pen between the index and middle fingers as requiring less effort than the ordinary way. But here again comes in the personal equation; this is an extremely awkward position for some penmen, and consequently in their individual cases causes not less but more effort in writing. The stylograph, although it destroys a man's penmanship, causes less effort perhaps than any form of steel pen, and may therefore be recommended as a prophylactic agent to those who are threatened with this affection.

The curative treatment for writer's cramp demands one essential element which unfortunately cannot always be obtained for it—the element of rest. It is the almost universal judgment that rest is the most important thing that can be prescribed. According to Solly, two months' entire rest will cure the disease if this be recognized early. Some allowance may be made for this writer's optimistic use of the word *cure.* As said already, I am inclined to believe that scrivener's palsy once established is almost sure to return if the same kind and amount of labor is put upon the arm as that which originally caused it. Still, without complete and often prolonged rest nothing satisfactory can be obtained; and with it a degree of endurance can often be established that will permit a person to do a fair share of work in comfort. Intermittent rest, or intervals of rest, is important; and an allotment of work, not too great for the writer's strength, should be made for him in his daily routine. It is better for a person to be able to do some work than none at all. Unfortunately the demands of commercial and industrial life usually do not recognize any such humanitarian considerations.

Electricity is no doubt beneficial in those cases in which rest can be obtained. I am rather doubtful of its ability to give relief in cases in which hard work is persevered in. In the absence of reactions of degeneration the faradic current may be used, but my preference is for the galvanic. The faradic current simply exercises, and possibly may even harass, muscles that are already over-worn; whereas the galvanic current, given by gently passing one sponge, now with one pole and now with the other, along the various muscles, probably exerts a much more powerful nutritive and alterative action upon them.

Wolf's method of gymnastics and massage has been lauded as of great value. Its originator was not a physician but a penman, living in Frankfort. It consists of active and passive movements of all the muscles of the arm and forearm, and of massage. Wolf directs, according to Lewis, rather arbitrarily it seems to me, a certain number of these active movements. Massage is accompanied with percussion of the muscles.

Drugs are of almost no avail in writer's cramp. Except for tonics, and these only for their general strengthening effects, it is difficult to see how anything can rationally be claimed for any of them. The bromides and all sedative medicines are absolutely contra-indicated for pure writer's cramp. The hypodermatic use of atropine seems to me to be a treatment of doubtful promise. Strychnine in this manner, in cases in which the element of fatigue is well marked, would probably be of service.

Local applications, except as adjuvants to massage, are of but little value. Hot and cold alternating baths may be tried with some hope of temporary relief.

Surgery has been appealed to in these cases. Stromeyer cut the tendon of the long flexor of the thumb with good result, but this result was probably due to the mere fact that the operation put the muscle at rest for a period of time. This treatment is to be recommended with caution. Nerve-stretching has failed.

Mechanical appliances for supporting the hand have been devised by surgeons and others, but their description cannot be given here.*

Telegrapher's Cramp.

In 1875 Onimus, in a communication to the Société de Biologie, called attention to a form of functional spasm, peculiar to telegraphers, which he called the *mal télégraphique.* This was apparently the first note that appeared in medical literature on an affection which is now unfortunately far from uncommon. In 1882 Robinson (*British Medical Journal*, November 4th, 1882) described very briefly four cases, but the disease still appeared to be considered one of "the rarest of medical curiosities." In 1884 Fulton (*Edinburgh Clinical and Pathological Journal*, February 2d, 1884) wrote an admirable paper on this affection, in which he analyzed the physiology of the movements involved in using the Morse instrument. Since the appearance of these initiatory papers the disease has steadily increased with the increased use of the telegraph, and it is now quite universally recognized.

Telegrapher's cramp is practically the same disease as writer's cramp, and as the latter has already been described in detail it is merely necessary here to refer the reader to that description for general information. There are some special points, however, in etiology and symptomatology which demand separate notice.

Onimus in his original paper stated that the Morse instrument was the one that caused this affection. This seems to have been confirmed by subsequent observers. This instrument, however, it must not be forgotten, is almost universally in use, and therefore is bound to receive most of the blame. It certainly appears, however, as must be acknowledged, to be particularly well fitted to cause this disease; in order to understand the reason for this it is necessary to consider its mode of action.

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* Debout (Bulletin de Thérapeutique, 1860, 327) describes and illustrates some of these devices.

The transmitting instrument, so familiar an object to all, has a key, which consists of a lever surmounted at one end with a knob. By depressing this knob a circuit of electricity is established which instantly acts upon the receiving instrument at the other end of the line. This receiving instrument contains a coil of wire enclosing a bar of soft iron. Every time the circuit is made by depressing the key in the transmitting instrument this bar of soft iron is magnetized by the current flowing through the coil; it therefore attracts an armature, fastened at one end of a lever, the other end of which holds an inscribing point (or an inked wheel, according to some) by which the appropriate characters are marked on a slip or ribbon of paper which is kept moving by clock-work.

The Morse alphabet is composed entirely of dots and dashes. It is as follows:*

A	B	C	D	E	F	G	H	I
. —	— . . .	. . .	— . .	.	. — .	— — .	. . . .	. .
J	K	L	M	N	O	P	Q	R
— . — .	— . —	—	— —	— .	. .	. . . . .	. . — .	. . .
S	T	U	V	W	X	Y	Z	&
. . .	—	. . —	. . . —	. — —	. — . .	. . . .	. . . .	. . . .

1	2	3	4	5
. — — .	. . — . .	. . . — .	. . . . —	— — —
6	7	8	9	0
. . . . . .	— — . .	—	— . . —	—
.	,	;	:	”
. . — — . .	. — . —	. . . . .	— . — . .	. . — . — .
$	’	(	-	
. . . . — . .	. . — — . . — . .	. . . . . — .	. . . . . — . .	

It may be seen that some of the letters of this alphabet contain more dots than others. Thus the letter H consists of four dots; S of three dots; X of a dot, a dash and two dots; V of three dots and a dash; U of two dots and a dash. Each of these dots and dashes requires a separate and distinct pressure on the transmitting key. The pressure must be a delicately coördinated act, causing just the right contact, repeating it rapidly, once for each successive dot and dash, and continuing it longer for each dash than for each dot.

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* This alphabet, a copy of which has been furnished me by Mr. Dunn (who is referred to later), is the only one, according to him, used in this country. It differs in many of its characters from the alphabet given by Fulton (*loc. cit.*) which is presumably the one used in Great Britain.

The telegraph operator usually holds the key with his thumb and fore and middle finger (Fig. 5). The basis of support is the elbow, and most of the movement is by the extensor and flexor muscles of these fingers and of the wrist. In addition, the adductor or opposing muscles of the thumb are in constant action. If now it is considered that each individual dot or dash requires a down stroke of the fingers upon the knob, followed by an upward movement before the next down stroke can be made, and that with the exceptions of the letter E, which is a single dot, and T, which is a single dash, every letter consists of more than one dot or dash (some characters consisting of as many as four and even more), it can readily be understood what a rapid, sustained, and coördinated movement is required for steady work. The word "telegraph" for instance contains nine letters, but to make them with the Morse instrument requires twenty-one separate dots and strokes; the word "occupation" contains ten letters and requires twenty-five distinct impressions.

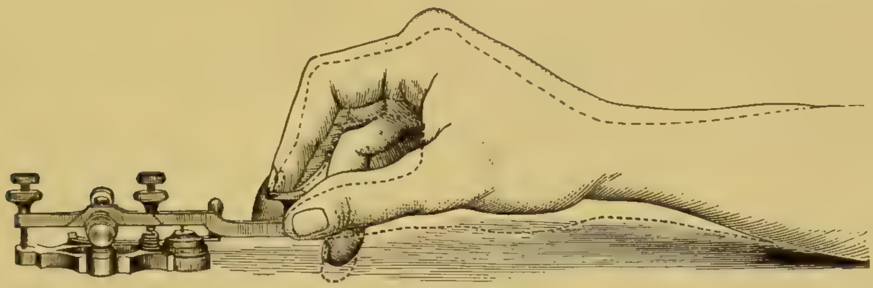

FIG. 5.—Normal Position of the Hand for Telegraphing. (After Lewis.)

According to Fulton's analysis of these movements (*op. cit.*) they consist of flexion and extension, chiefly at the wrist, but slightly at the elbow and partly at the metacarpo-phalangeal joints of the index and middle finger. The muscles exercised are the extensors of the carpus and of the fingers, especially that part of the common extensor that controls the movements of the fore and middle finger, and the special extensor of the index finger. The extensors of the thumb also are involved. The downward movement is made by the flexors of the wrist and of the fingers, especially, as in the case of the extensors, by those portions of the common flexor that act upon the fore and middle finger. There is also a constant but variable contraction of the adductor, opponens, and flexor muscles of the thumb. The interossei and lumbricales also are brought into use, as are also in a minor degree the biceps, triceps, and even some of the shoulder muscles.

The number of distinct but similar muscular movements made by a telegraph operator during a busy day's work is difficult to be estimated. Fulton calculated that an operator makes between thirty and forty thousand contractions per hour, and on busy circuits he may be engaged constantly for eight hours. One man told me that he had worked so long as fourteen hours in one day. This, however, is probably far in excess of the average. The amount of work varies of course in different offices and upon different kinds of matter. Transmitting speeches and long documents requires a sustained effort which is exceedingly hard on the muscles. In many offices the operator uses his hand for writing almost if not quite as much as for transmitting, and it is not uncommon for him to have the same difficulty in writing as in telegraphing.

Mr. George W. Dunn, traffic chief of the Postal Telegraph Cable Company in Philadelphia, informs me that telegrapher's cramp is not uncommon among operators, and that it is rather increasing in frequency. He thinks that it is aggravated by drinking habits. Stimulation sometimes enables a man to overcome the difficulty temporarily, and hence some men may be tempted to indulge, but afterward the disorder is worse. According to Mr. Dunn the majority of those who have it are addicted to some use of alcohol. Speaking of the affection itself, he says that he knows of one man who is obliged to strike the key with his thumb instead of taking hold of it in the usual way.

Onimus makes a somewhat similar observation to this last by Mr. Dunn. He refers to an operator who used successively his thumb, his index, and his middle finger; each of these fingers retained its normal action for two or three months, but one after the other they were eventually seized with spasms. Finally the man used his wrist, which after a time refused likewise to do the work.

With reference to drinking habits, I can only say that I have known of one man, grievously affected with telegrapher's cramp, who neither drank nor used tobacco, and who was in every way, except for his fatigue neurosis, in robust health. I do not doubt that alcohol and tobacco will aggravate this affection, and perhaps even predispose to it, but I wish to caution against the mistake of looking upon these poisons as prerequisites to the disease, or upon all telegraph operators suffering with cramps as addicted to these excesses.

It is not probable that sex has an appreciable influence in causing this affection. I have made no observations which would lead me to suppose that it is more common among women than among men.

The onset of the affection is sometimes rather sudden, even in men who have worked for years without observing any symptoms of it. The first thing noted is usually a difficulty in making some particular letter, especially one of the letters that have several dots in succession. In the act of making these dots in rapid succession the hand suddenly becomes cramped and refuses to do its work. One man may have difficulty with one letter, another with another. Because of these difficulties one letter may be made when another is intended, thus U (- - —) may be made for V(- - - —), or S (- - -) may be made for H (- - - -). These mistakes in transmitting of course entail loss of credit and may even lead to serious results; and the knowledge of these facts reacts disastrously on the mind in some cases, causing worry and loss of self-confidence which do not improve the operator's skill. Onimus refers to certain neurasthenic symptoms, such as loss of memory (or of attention?), and hyperæsthetic cervical spine which are probably directly traceable to these mental states.*

The type of telegrapher's cramp is almost exclusively the spasmodic. Robinson, to be sure, includes among his cases that of a young woman who suddenly lost power in her arm after a disappointment and who in consequence could not use her telegraph key. The case has every appearance, although badly reported, of being one of hysterical brachial monoplegia, happening by mere coincidence in a telegraph operator. Tremor is seen in some of the cases, but in all those in which I have seen it there was a distinct history of the excessive use of alcohol and tobacco. It is not sufficiently distinct in any of these cases to constitute a "type"; in fact it is better to avoid the error of trying to see distinct types in this affection. The spasmodic action, just as was pointed out in the case of writer's cramp, causes incoördination and jerky to-and-fro movements, but these cannot be regarded as tremors. The tremors that I have seen have persisted between the attacks, when the hand was not at work, and were suggestive of some slight toxæmia.

As the spasmodic action is not unlike that which is seen in writer's cramp it is scarcely necessary to do more than describe it briefly. It seizes the patient's hand while he has hold of the key with his thumb and fore and middle finger. It is not so apt to be a flexor as an extensor spasm. The extensor muscles become firmly set and effectually pull the hand away from the table, and the fingers even away from the instrument. This action is perfectly shown in the illustration (Fig. 6) from the photograph of a patient whose arm is thus spasmodically drawn up by over-action of the extensors. This man, whose case is reported further on, is only able sometimes to manipulate the key with his thumb. This spasm as a rule is not painful; there may, however, be painful spots in the arm, and neuralgic pains in some areas. It is not associated with tactile anæsthesia, but in some cases there is a subjective feeling of tingling and numbness. As in the case of writer's cramp, I should not expect to find true muscular atrophy and, least of all, the reactions of degeneration. I am not aware that these have yet been reported. It has at least not been my lot to see them, and in any case in which I should chance to find them I should look for some deeper-seated cause than is found in any of the fatigue neuroses.

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* As evidences of the facility with which mistakes can be made by substituting one letter for another, Onimus gives a few instances of the transformation of his own name (viz., Oteimus, Otomus, Obmus, etc.) and says that the word *référé*, by defective transmission of the *e*, alone, can be mutilated in four hundred and forty-seven different ways. (Comptes Rendus de la Société de Biologie, 1878, p. 94.)

The following are illustrative cases which have come under my notice:

A. J. W., æt. 36; white; male; of American birth. He has been a telegraph operator for sixteen years. The first symptoms of cramp were noted after he had been at work between six and seven years. At this time he drank heavily, and used tobacco to excess. The first difficulty he had was in making the letter B (—---); his hand would act involuntarily and would make four dots (the numeral 8). This was an entirely involuntary movement. Then later cramp-movements came on whenever he tried to use the instrument. His special difficulty now is in making the letter P (-----), which causes a cramp because of the rapid succession of dots. He feels some pain in the shoulder. He has taught himself to work with his left hand for the last four years. He can now work as fast with his left hand as he formerly could with his right, and has thus far had no cramp in it. He has never had difficulty in writing or in any other movement.

Examination in this man's case demonstrates that there is no muscular wasting; that all the muscles respond normally to electricity; that there is no tactile anæsthesia, but that there is perhaps a slight paresis, especially for associated movements.

Points of especial interest in this case are the facts that the patient, according to his own statement, has been able to train his left hand to work as well as his right, and that after four years there is as yet no symptom of the affection in it.

With reference, however, to the claim, which some of these men make, that they can work as well with the left as with the right hand, I always make some allowance for the natural exaggeration into which a man may be led on such a point. These operators are rather sensitive about their defect, and some of them are not a little despondent about it. When they find that they can train the left hand they rapidly regain hope and confidence, and under such circumstances they are apt to think that they are as well as ever. I fear that there is always a doubt about the ability of the left hand to do so much and so good work as the right; and that there is a strong chance that it will in time go the same way as its fellow.

A second case is as follows:

B. C., æt. 22, white; male. He has been a telegraph operator for eight years. He had worked so long as fourteen hours a day, just before this trouble came on. On some days he would write all day, on other days he would transmit all day. (The writing is done, of course, when receiving messages; the work on the key when sending them.) The affection came on rather suddenly, *i.e.*, in one week, getting worse until he could neither write nor transmit. In his case the affection involves both writing and using the key, and also using the type-writer. This, he says, is not unusual in these cases. He uses his left hand now, but acknowledges that he cannot send so fast as formerly with his right. He has no symptoms of the affection in his left hand as yet. When he attempts to work the key with his right hand the limb is the seat of an involuntary movement, by which the hand is pulled up and away from the table. He writes in a slow and constrained manner. He says that he has a numb feeling in the hand. He worried about his condition until he found that he could educate his left hand. He has no pain.

Examination of this man's arm shows that the muscles are hard and strong and that there is no atrophy. There is slight tremor, which may be due to tobacco, of which the patient uses too much. There is no fibrillation, nor any tactile anæsthesia. The reactions to both the faradic and galvanic currents are normal. The man is apparently a beer-drinker.

A third and last case is as follows:

H. W., æt. 36, white; married. He has been a telegraph operator for twenty-one years. He has had difficulty in using his hand for fourteen years. The affection involves only the right hand, and interferes with writing as well as with transmitting. Writing did not become affected until three years after the transmitting was first interfered with. The patient has pain even when not using the arm, especially at night. If the hand is outstretched the pain is increased, but if the fingers are firmly flexed, or something large is held in the hand, the pain is relieved. On busy days he works very hard, sending so many as three hundred messages in one day. He does not use alcohol or tobacco.

The examination of this patient, an illustration from a photograph of whose hand in action is shown in Fig. 6, gives the following results: When transmitting, the hand gradually becomes stiff. The cramp is painful. It predominates in the extensors, pulling the hand away from the table; the little, ring, and middle fingers especially are pulled up (see Fig. 6). The patient manages to keep hold of the key, as is seen, but the muscles of the arm and hand are firmly set and hold the limb in an iron-like rigidity. Although the action of the extensor muscles predominates, the flexors also are firmly contracted, at least those that act upon the thumb and the forefinger. In the forearm, the muscles stand out prominently because of the cramp-like action. A slight incoördinate action of the hand is caused by the spasm.

There is no anæsthesia in the hand or arm; neither is there tremor, fibrillation, nor atrophy. The electrical responses have not been tested, but from the appearances of the muscles, which are full and firm, there can be little doubt that these would react normally.

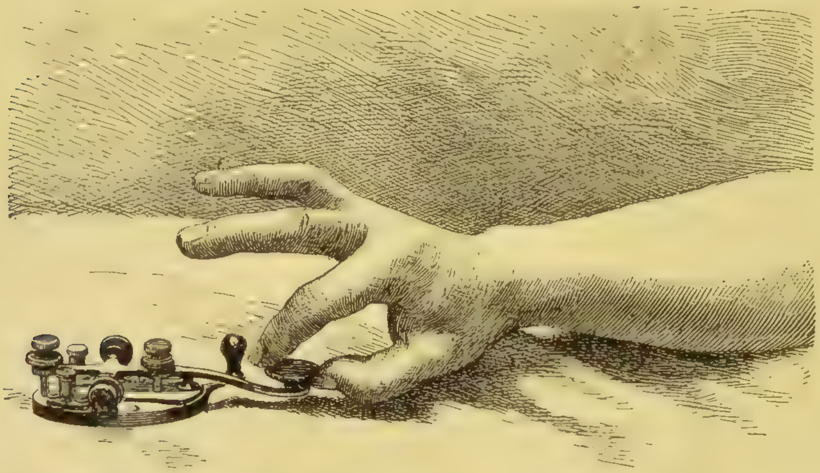

FIG. 6.—Telegrapher's Cramp. Spasmodic action of the extensors tending to draw the hand away from the button.

This last is one of the few cases of fatigue neurosis that I have seen in which the cramp has been painful. As an evidence of the involuntary character of this cramp, it is worthy of note that the patient was obliged to work the key for some time before the spasm could be induced for the purpose of being photographed. It seemed to me, as I watched the man's efforts, that the consciousness of being observed and of sitting for this special purpose induced a mental state which acted as an inhibition upon the peculiar muscular affection.

The *treatment* for telegrapher's cramp must be conducted on practically the same lines as those laid down for the similar affection in writers. Rest is the most important element of this treatment. It is remarkable, however, how long some of these operators continue at work in spite of the disease. They seem to have some advantages over scriveners, due to the fact that they can manage better by makeshifts to use their keys than writers can to use their pens. They are able also with more facility to educate the left hand to work upon their instruments than writers are able to learn to write with this hand. The explanation for these differences probably lies in the fact that the movements for telegraphing, while exceedingly rapid and of very fine adjustment, are yet not nearly so complex and specialized as are those for writing. The former are after all merely up and down movements of definite range, calling into operation comparatively few coördinated muscular activities, while the latter are of much greater range and variety, concerned as they are with tracing on paper letters and figures of great complexity of design. Too often, to be sure, the telegrapher becomes the victim of scrivener's palsy, when of course his lot is doubly hard, and when the plan of treatment already suggested for writer's cramp applies to him.

Telegraphers are not able to seek relief readily, as do writers, by altering the shape and style of the instrument with which they work. They are obliged to use the one key. I have never heard that they have sought or been able to find relief by changing this key in any way. It might be in some cases that if the knob or button were enlarged the cramp would be modified; but this hardly seems probable, because the spasm affects chiefly the extensor muscles of the wrist and of all the fingers, not merely of those fingers that grasp the key. Still, I should like to see this method thoroughly tried, and would suggest the use of a very large knob, even as large as a door knob, made of some light material or papier-maché, upon which the patient's hand could rest easily without much effort in flexion.

Electricity and tonics may be recommended, just as they were for writer's cramp. They are serviceable in conjunction with rest. Galvanism is the preferable current.

Massage and the Wolf treatment with active and passive movements are also worthy of trial.

Finally good habits and hygiene are essentials. There can be no doubt that the excessive use of alcohol and tobacco will aggravate the tendency to and the severity of telegrapher's cramp. Unfortunately these men often are overworked, and their surroundings and associations lead them perhaps into temptation, but it ought to be impressed upon their minds that stimulation is a temporary and bad crutch.

Miscellaneous Forms of Fatigue Neuroses.

According to Hall (*op. cit.*) the Sheffield grinders are occasionally affected with a form of palsy due to fatigue in grinding.

In 1869 Dr. Frank Smith, of Sheffield, England, described (*Lancet*, March 27th, 1869, p. 427) some cases of paralysis occurring in Sheffield cutlers, under the rather misleading title of "hephæstic hemiplegia" or hammer palsy. He seemed to think that this affection had points of resemblance to "scrivener's palsy," from which disease, however, it differed by the absence of spasm and the extension of the paralysis. An attentive study of Smith's very meagerly reported cases fails to discover anything unique or remarkable in them. Several of them were cases simply of hemiplegia from brain disease (embolus or hemorrhage, apparently) happening by chance in men whose work required them to use a heavy hammer. In some of these patients there was not only paralysis of the arm and leg on one side but involvement also of some of the cranial nerves, as, for instance, the third and seventh. In a few of these cases, however, there was apparently a true fatigue neurosis. Thus one man, a table-blade striker, who had used a heavy hammer for twenty years, had weakness, wasting, and numbness of the right arm; he had also neuralgic pain over the shoulder. Another man, a file-forger, had partial loss of power with wrist-drop in the right hand and arm; he could not grasp with his right hand.

Arlidge says (*op. cit.*) that engravers have contracture of the little finger from its constantly cramped position; also semi-luxation of the thumb backward from pressure. The fingers are liable to a spasmodic action, analogous to the cramp of scriveners and telegraphers. Layet says that engravers have also anæsthesia of the skin of the hands and forearms, and atrophy of the interosseous muscles. I am informed by a patient, who is an engraver of types, that the work is trying to the eyes. He himself has never experienced any cramp in the hands, although he works on very fine letters and designs.

Performers on the piano-forte suffer with a spasm of one or more of the fingers. Erb (*op. cit.*) says it is not uncommon among professionals, especially women. It may be accompanied with the same form of neurasthenic spine as that noted by Onimus in telegraphers. This is experienced especially in the cervical spine and between the shoulders. I think this neurasthenic spine is not uncommon in music-teachers; I have seen several instances of it. It might readily be associated with spasm in the overworked muscles of the hands and arms.

Richardson ("Diseases of Modern Life") speaks of a player on a violoncello who suffered with paralysis of those fingers of the left hand that were exercised on the strings of his instrument. Erb says that this painful exhaustion, stiffness, or spasm may occur in the right as well as in the left hand of these performers.

Arlidge refers briefly to the makers of artificial flowers as being affected with a fatigue neurosis.

Bricklayers are reported, especially in England, to have a spasm or cramp occasionally from prolonged grasping the trowel. It is said to have been a rule of the bricklayers' union that the trowel should never be removed from the right hand. The constant and prolonged strain caused cramp (editorial in London *Lancet*, 1875).

Erb calls attention to sewer's spasm, which affects tailors and seamstresses. As soon as they begin to sew the fingers become cramped, and their work is effectually interfered with. The affection is practically identical with writer's cramp in its main features, differing only in cause and in the particular muscles involved. Erb draws a distinction between this spasmodic action and the form of tetany that has been described under the name of "shoemaker's spasm," but observers ought to be careful to notice the points of similarity which the two have as well as the points of difference.

Basedow described a milker's spasm. This is said to be met with among the cowherds of the Tyrol.

Type-setters occasionally have a form of spasmodic affection in the muscles of the hands and arms, but it is rare.

The following case, at present under my observation in the Philadelphia Hospital, presents some points suggestive of the professional muscular atrophy that has been described by Onimus and others (see *supra*).

D. McC., æt. 53, white, with a good family history, a stonecutter by trade, had been doing very hard work at bridge-building. He had stood much in the water. He confesses to a free use of alcohol and tobacco. In his work he used a heavy hammer. After severe and long-continued work, in all kinds of weather, he noticed that his shoulder was wasting. This wasting progressed downward, involving the arm and hand until the patient was no longer able to work. The nutrition of the body remained good until an attack of dysentery reduced him; after which the muscular atrophy extended to other parts of his body. At the present time the man has muscular atrophy involving the shoulder-girdle, arms, forearms, hands, back, chest, thighs, and calves. There are no contractures, but a slight fibrillation can be noted occasionally. The myotatic irritability is increased; the knee-jerks are present but rather sluggish. The biceps jerks are present. There is no tactile nor thermal anæsthesia. The wasted muscles respond actively to faradism, and there is no reaction of degeneration to galvanism.

In this case it is to be noted that the affection began in the shoulder, extending to the arm, in a man who wielded a heavy hammer; that it was gradual and progressive, and that, although it has occurred in a man who was much exposed to wet and cold, and who drank too much alcohol, it nevertheless is not a multiple neuritis but preserves the type of a muscular atrophy.

MINER'S NYSTAGMUS.

This curious affection undoubtedly belongs to the group of the fatigue neuroses. It has been investigated, among others, by Dransart and by Snell. The latter's paper (Transactions of the Ophthalmological Society of the United Kingdom, Vol. IV., 1884, p. 315) is an especially valuable one and evidently gives the correct pathology for the disease.

It has been known for a long while that miners, especially coal miners, are subject to a nystagmus, especially when the eyes are directed either obliquely upward or above the horizontal plane. This nystagmus may be either lateral or rotatory, or sometimes both. The effects of this oscillation of the eye-balls vary. In some men the tremor is borne without inconvenience for years, but in others it causes vertigo and dancing movements of the images of objects seen, as lights, etc. I have no doubt that in some men it causes anxious mental states and tends to induce neurasthenic symptoms; especially in conjunction with the anæmia that not uncommonly affects these workmen. According to Snell, many of those affected are able to bring the eyeballs to a standstill by looking steadily before them; but lateral and especially oblique upward movements cannot be borne long without pain and discomfort and a return of the oscillation. Rapid walking or running or elevating the head suddenly from a stooping position will bring on the affection. Cases vary in severity. In some the movement is constant, in others it appears to be intermittent, or, as Snell says, latent. In these latter the movement requires to be excited in one of the ways mentioned before it can be detected.

This affection is not associated with evidence of any deep-seated disease of the brain or nervous system. The vertigo which sometimes accompanies or is caused by it can readily be understood. This vertigo is not associated with nausea or tinnitus.

Various attempts have been made to explain miner's nystagmus. One theory is that it is caused by a strain of the muscles of accommodation and convergence caused by working in the bad and flickering light of the safety lamp. Dransart gives prominence to this strain, referring to the intimate relations that exist between the actions of the ciliary and internal rectus muscles. The miners themselves, it is said, have the firm opinion that the nystagmus is caused in some way by the safety lamp. Snell's explanation, however, is the most reasonable of all. According to this observer, who descended into a coal pit in order to pursue his investigations, the affection occurs in those miners only who are employed in excavating the coal by digging underneath the seam. In order to do this they are obliged to lie on one side in a narrow space with their eyes cast in an upward and oblique direction. This is well illustrated in the accompanying pictures from Snell's paper (Figs. 7 and 8). This position entails great strain upon the levator, superior rectus, inferior oblique, and external rectus muscles of one eye and upon the same muscles in the other eye, except that the internal instead of the external rectus acts. In other words the work requires a sustained and coördinated movement of a number of delicately adjusted and associated muscles, which entails a strain upon these muscles and results in an oscillatory or spasmodic movement in them. Hence the disease is strictly analogous to writer's and telegrapher's cramp.

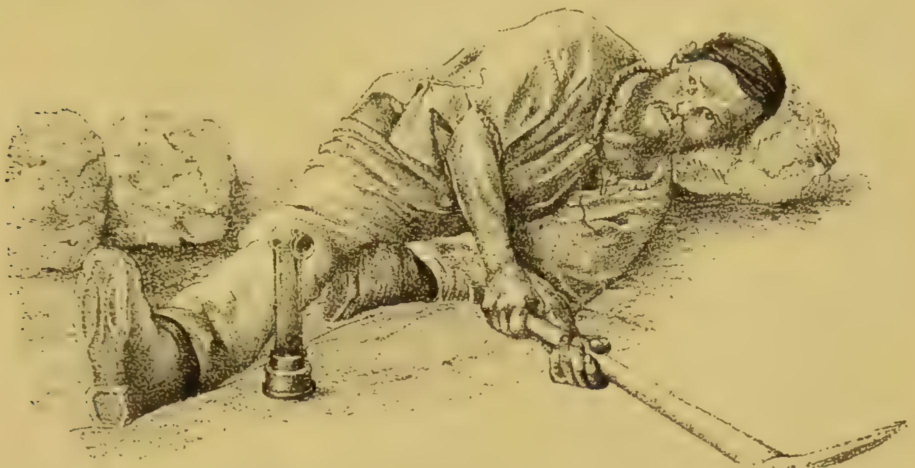

FIG. 7.—Miner's Nystagmus. Position of the miner while at work. (After Snell.)

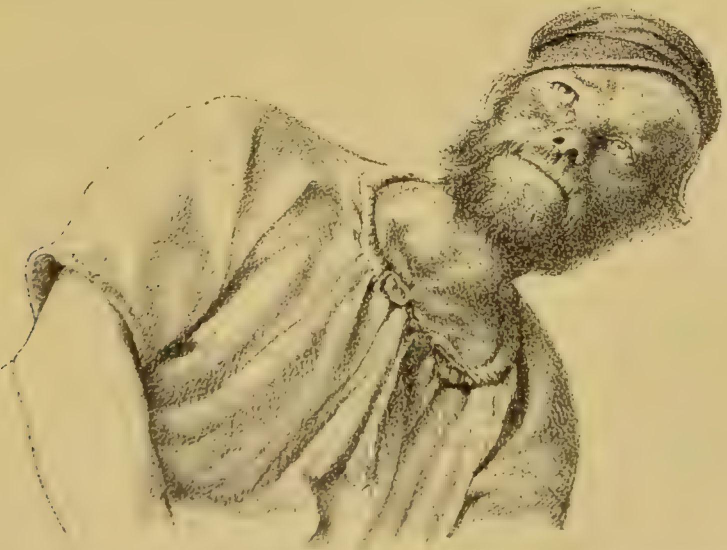

FIG. 8.—Miner's Nystagmus. Constrained position of the eyeballs while the miner is at work. (After Snell.)

In confirmation of his view Snell found that workmen in the mines who did not do this particular kind of work were not afflicted with nystagmus.

The *diagnosis* requires that the disease be distinguished from disseminated sclerosis and from the organic diseases of the eye and brain that cause nystagmus. This is comparatively easy to do when the history of the case and the patient's occupation are considered, and when it is observed that no involvement of other muscles and no defects of brain or speech are present.

The *treatment* is by rest; this means usually in these cases a change of occupation which, by relieving the peculiar strain, is really equivalent to rest.

EFFECTS OF THE SEWING-MACHINE.

A lively controversy about the effects of the sewing-machine upon health prevailed at one time. Some very sensational claims to the effect that this useful machine was disastrous to both the health and the morality of women were seriously put forth; claims which if true would, as an eminent French writer has said, have abolished its use from industrial life, but which now are recognized as among the amusing extravaganzas of medical literature. On the other hand, claims almost as preposterous were made that the sewing-machine was a boon to the health and happiness of overworked women. As is usual, the truth lies probably between these extremes.

Vernois in 1862 was probably the first to call serious attention to what he considered some of the evil effects of the sewing-machine. He claimed that it caused irritation of the sheaths of the tendons of the extensor and flexor muscles which induced cramps and even paralyses. Some of these observations were undoubtedly correct and will be referred to later. Ord made a temperate report (Sixth Report of the Medical Officer of the Privy Council, 1863) in which he concluded that on the whole, with the increased pay obtained with their use, the working of these machines was not very injurious, although attended with some evil results as in the case of all overwork. In 1866 Guibout made his startling declaration that the peculiar movement required for working these machines caused genital irritation, and hence not only induced uterine disorders but led to vicious practices and immoral sexual indulgence. This extravagant claim was based on a ridiculously inadequate number of observations, and made far more impression at the time than it deserved. It was re-echoed by Down (*British Medical Journal*, 1867, Vol. I., p. 27), in England in the following year, but without much effect. The same may be said for Fournier's single observation of abolition of sensation of the sole of the foot, which he attributed to the contact of the foot with the treadle, but which was in all probability an hysterical anæsthesia.[*]

The most scientific and carefully prepared papers on the effects of the sewing-machine on health are those by Espagne, Decaisne, and Nichols. The truth about the sewing-machine, so far as it can be gathered from these observers, is about as follows:

In the motor system Decaisne, whose observations were made on 661 women, found fatigue, vague muscular pains, and pains in the loins.[†] These pains disappeared usually with rest. Some women had special kinds of pains in the thighs, and sometimes cramps. In not one patient was there observed anæsthesia of the soles of the feet. The lumbago and pain in the thighs were not peculiar to the women who used sewing-machines, but were common among other sewing-women in Paris. These pains were less common among those who had worked for several years than among novices. In women at or approaching the menopause, however, these lumbar pains were more frequent. The general conclusion is that these pains do not occur in the ordinary household use of the sewing-machine, and that in women who work for long periods they disappear in general after a certain time. Hence this observer believes that these sewing-women become accustomed, as a rule, to bear the muscular strain.

Nichols also observed that cramps are among the symptoms of these cases. These cramps do not seem, however, in any cases to assume the importance that they do in writer's and telegrapher's cramp; in fact, they hardly constitute the same type of affection, because they tend to recovery rather than to chronicity.

This alleged tendency of women to establish a tolerance for the fatigue of working the sewing-machine may be true within certain limits, but it seems to me that there may be numerous instances in which the excessive or disproportionate use of these machines leads to a degree of nervous prostration. Much of course will depend upon other factors, as the ventilation of shops, food-supply, and personal hygiene.

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[*] The tendency to attribute all sorts of diseases to a particular occupation (on the principle of *post hoc ergo propter hoc*) is well shown in the case of the sewing-machine. Thus Guelliot traces two cases of locomotor ataxia to its use. The same tendency is strikingly shown in Nichols' paper, referred to in the text, in which are embodied the opinions of many general practitioners in Massachusetts who ascribe uterine diseases indiscriminately to this machine.

[†] The original French word is *reins*, which Nichols erroneously translates kidneys. There is no evidence of any affection of the kidneys in these operatives.

Decaisne could not find evidence that digestive disorders were more rife among these work-women than among others. He reached practically the same conclusion with reference to disorders of the respiratory organs.

As for the nervous system, Decaisne says that the shock or jar caused by the noise and motion of the machines has practically no permanent effects. It may be operative upon novices, but these soon become accustomed to it.

This same careful observer scouts utterly the assertion that working on these machines causes genital irritation. He says moreover that a rigorous inquiry disproves the assertions that the sewing-machine causes metrorrhagia, miscarriages, peritonitis, or leucorrhœa, and he asserts that when these occur they are coincidences.

In short, Decaisne concludes that work with the machine, within reasonable limits, is not more injurious than work with the needle.

Nichols' conclusions are not so optimistic as those of Decaisne, but his methods and mental attitude do not appear to have been quite so scientific and critical as those of the French writer. His conclusions are drawn from the hasty reports of a large number of medical practitioners, and from the testimony of mill-proprietors or foremen, many of whom probably had made but few and not always accurate observations. He thinks that indigestion, muscular pains, diseases peculiar to women, and general debility—by which latter term he probably means neurasthenia—are caused by work on the machine. I think that in the last conclusion he is probably right. Excessive use of the sewing-machine, like any other form of overwork, may cause neurasthenia, but in this respect it is not peculiar or distinct.

The synchronous rather than the alternating pedals are recommended as less liable to cause fatigue and genital irritation. But the most important prophylactic means, and one which it is only necessary to mention here, is the substitution of steam for foot-power. In most large factories this probably has now been made.

NEURASTHENIA FROM OVERWORK.

In concluding this division on the "Effects of Injurious Exercise" it seems proper to discuss briefly the subject of neurasthenia as caused by overwork. It is not an easy task to describe this affection in short space, but, considering its importance, it cannot be ignored entirely in this connection, because among its well-recognized and ever-active causes is the strain of excessive labor. There is nothing essentially new about the subject except its title. A tendency prevails, however, at present to include under this term a variety of symptoms and states, some of which belong properly to other conditions, especially of the nervous system. Thus the term is made by some to include symptoms which actually belong to hysteria; but this error is not so likely to occur in reference to overwork as to trauma. The term is really a useful one and undoubtedly has come into medical literature to stay. Hence the necessity for securing as strict a definition of it as possible. Löwenfeldt, among recent writers, is disposed to confuse the affection with hysteria, but this identity will not be recognized, I feel sure, by American writers, as the stigmata of the great neurosis come to be better apprehended.

Neurasthenia is usually defined as a condition of *irritable weakness*. Its etymological meaning, viz., weakness of the nervous system, is rather too restricted in scope, because the nervous system is not alone affected in this state. It is a condition apparently of malnutrition, in which many tissues of the body share, and it acquires an identity by the rather characteristic grouping of the manifold symptoms thus induced.

Tissot, in his classical essay on the diseases of literary and sedentary persons (*op. cit.*), gave really the first systematic description of this condition as it is observed in overworked scholars, professional men, etc.* Many of his descriptions answer admirably for the more modern aspects of the disease. Ramazzini, before Tissot, had devoted a chapter (*op. cit.*) in his sketchy way to the same subject. The literature of the past few years is very full, one of the best monographs being the Toner lecture by Dr. Charles K. Mills. Dana, in his recent work on nervous diseases, claims that overwork does not cause neurasthenia, provided there is no great worry; but it seems to me that this saving clause rather begs the question. It must be evident to one who studies the hygienic surroundings of many of our laboring classes that overwork is often associated not only with worry but also with bad ventilation, poor food, and defective domestic and personal hygiene. This combination of causes is potent to induce neurasthenic states. I do not know of any more favorable conditions for its causation than can be found in some of the overheated and ill-ventilated cotton mills described by Arlidge, in which the operatives are cooped up and overworked while subsisting on poor and scanty food and consuming an excess of tea. As already seen in this article, special conditions among workmen and women may cause it. Thus a neurasthenic taint, if I may so call it, is seen in some cases of writer's and telegrapher's cramp, and, according to Nichols' description, is found in women who work to excess on the sewing-machine. I have often been struck, during my researches for the preparation of this article on the diseases of occupations, with the numerous facts that have come to my notice tending to convince me that one of the most common effects of overwork and poor hygiene in industrial life is an ill-defined condition of neurasthenia. It is not possible here to do much more than merely to indicate this general fact and to call the reader's attention to the necessity for recognizing this element when he is brought in contact with the laboring classes. To enter into further details would make it necessary to traverse much ground which has already been passed over.

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* Tissot's original essay was in Latin, his dedication bearing the date 1766. This essay was translated by its author into French, and a translation appeared a few years later in English. This latter translation is the copy of the book from which I quote.

I would call special attention, however, to the prevalence of neurasthenia among brain-workers, the more so that I have not devoted any special chapter in this paper to their ailments. It is found particularly, in my observation, among professional and business men who, in addition to hard work, have borne the burden of anxiety and various physical excesses. I do not think that it is prevalent, however, among physicians. In young women who support themselves by brain-work, as school and music teachers, a mild grade of neurasthenia is not uncommon. It is more common in them than in another class who combine longer hours and perhaps poor living with some responsibility, as clerks and shop-girls. I think that it is rather more prevalent among young women than in young men who take harder and more responsible positions.

In reference to professional men the observation of Tissot is still full of significance: "The diseases to which the learned are particularly exposed," says he, "arise from two principal causes, the perpetual labors of the mind, and the constant inaction of the body." Perhaps one reason why physicians are not subject as a rule to these diseases is that they do not experience the effect of this second cause.

The most characteristic *symptoms* of neurasthenia are purely subjective; they are the results of a mental state which is new to the patient and which perplexes and harasses him. He feels obscurely that a change has come over him, but he has difficulty in describing this change. He experiences a feeling of nervousness and of mental ill-health. His duties are a burden to him. He is easily worried and fretted and cannot apply himself with his old-time facility and spontaneousness. This irritable weakness of the mental faculties leads naturally to introspective attempts on the patient's own part to interpret the change that he has experienced. Hence exaggerated and erroneous ideas often take possession of him as to the causes, the tendency, and the gravity of his affection. Closely allied to the mental uncertainty, or perhaps caused by it, are the impulsions and fixed conceptions that sometimes appear. These, which have been classed properly by Regis, in his recent treatise on insanity, with neurasthenic phenomena, have been regarded too often by others as stigmata of incipient paranoia. These impulsions and conceptions are innumerable; sometimes they have an hypochondriacal tinge, as when they merge into apprehensions for the patient's health, and thus induce corresponding depression both of spirits and of health; at other times they partake of the character of the so-called intention psychoses, *i.e.*, they become genuine imperative conceptions propelling the patient to pseudo-volitional acts of great variety and complexity.*

Associated with this mental state are other nervous phenomena, as insomnia, headache, and pains in and along the spine. This irritable spine is quite common in the neurasthenia of the female sex. The pain may centre chiefly in the cervical spine and about the base of the skull, or between the shoulders; in some cases, however, it is most acute in the lumbar region. Other more obscure sensory phenomena are feelings of numbness in the extremities, and pricking sensations. Extreme lassitude and weakness occur, but genuine paralysis, as seen in hysteria, does not appear. A slight tremor and muscular instability may be seen.

The special senses, excepting sight, are not as a rule affected. Sight, or rather the musculature of the eyes, may be impaired. Weakness of this apparatus leads readily to eye-strain and its concomitant brow- or headache.

There is not unfrequently cardiac irritability, marked by rapid pulse on slight exertion; and vaso-motor weakness, shown by flushings, cold extremities, hyperidrosis, and urticaria-like eruptions as results of irritation of the skin.

To these rather common and characteristic symptoms many writers add a long array of others, which in truth are merely the various evidences of dyspepsia and imperfect elimination. The mental symptoms, which I have described, are to my mind the most characteristic phenomena of neurasthenia; they are the ones without which the affection does not exist. All others follow them as sorts of secondary phenomena. The temptation (and it is one that I wish to avoid) is to include too much in the description of this state, which, to say the least, scarcely any two writers as yet describe alike.

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* I am convinced that Regis is right in regarding many of these intention psychoses as neurasthenic phenomena. They are often recovered from in favorable cases, and do not lead necessarily, as some would have, to more profound mental deterioration. They cannot be discussed in the text. The reader is referred to Regis' work: "A Practical Manual on Mental Medicine," translated by H. M. Bannister, A.M., M.D.

The *treatment* for neurasthenia from overwork should be, first, by removing or modifying the cause as much as possible. In the cases of many professional men, as well as artisans and those depending on their labor for their support, this is a difficult thing to be accomplished. Much may be done, however, by wise advice; by the physician making himself thoroughly familiar with the routine of the daily toil, and then offering such suggestions as his judgment and the circumstances warrant. No general rule can be given for this. The advice must be adapted to the exigencies of each individual case. It is sometimes possible to secure a limitation to the amount of work, and to arrange for an improvement in methods of labor, and in the matter of sufficient rest and of relief from the more harassing details. Without some such change treatment with drugs will often be of little avail.

In the second place, treatment with proper diet and drugs is of great importance.

The digestion of these patients being often at fault, it is essential to select a good and appropriate dietary. With a weakened digestion, I think that it is not always well to recommend highly nitrogenous food, meaning by that a diet rich in meats. This can be avoided by giving milk and eggs, and custards made from these, which are more digestible than meat. Strong beef broth, or even tender raw or underdone beef and mutton, may be given, of course according to the case. Vegetable food ought to be given in a fair proportion. It is rather more important, in fact, to secure a proper quality, quantity, and preparation of food than to limit the selection of it too rigidly to certain articles. Most people have idiosyncrasies about, or at least preferences for, their food, and in many cases it is wise to consult these. If proper meal-hours are secured, and the food is properly prepared and then properly masticated, the physician and patient are usually on the right course with reference to diet.

Hydrotherapy is a most valuable means for treatment. A cold sponge bath, or in some cases an alternating hot and cold bath, is a powerful alterative and tonic. Sponging the spine is a source of great comfort and relief to patients with spinal neurasthenia. Sea-bathing does well for some few of these patients, provided they react well from it.

In my experience the most valuable drugs are strychnine, quinine, iron, phosphorus, the mineral acids, especially the hydrochloric, the bitter tonics, arsenic, and cod-liver oil.

The hypnotic and sedative medicines are of doubtful utility in most of these cases, and are prone to do mischief if they are not used with the greatest care and moderation, and only under the direct supervision of the physician. Phenacetin, antipyrin, and salophen may serve a purpose very rarely to relieve pain and nervous irritability, but they are to be given with due precaution that the patient does not become familiar with them and dependent upon them. The same may be said of the bromides. I think that chloral, the opiates, and cocaine should have no place in the treatment of neurasthenia.

Electricity is sometimes of great benefit, especially in cases of the neurasthenic spine. Women especially are subject to this weakness, and they often derive much good from the galvanic current. This is to be applied directly to the spine, or rather along each side of it, by stroking with the wet sponges. The direction of the current is of no significance. Faradism is not so good, but still it may be tried in some cases, its use being alternated with galvanism.

Change of scene, out-door life, gentle exercise, and the like, are beneficial, but in the cases of the classes of whom I am now speaking these advantages cannot always be secured so thoroughly as could be wished.

Special conditions require to be treated. A lithæmic state is sometimes present in these patients and should be met with lithia water, nitro-muriatic acid, colchicum, and saline aperients.

Eye-strain should be searched for and, when found, should be relieved.

Finally, too much cannot be said for moral encouragement, support, and control. The physician does not do his duty to these patients unless he gains their confidence, and impresses upon their minds with wise suggestions the belief that they are curable and that he can cure them.

Effects of Constrained Attitudes, Etc.

Closely allied to the subject of over-use of various parts is this of constrained attitudes. It may be said, indeed, that there is probably not a mechanical trade that does not leave some mark, scar, or callosity upon those following it. These are so numerous, and many of them so trifling, that it is neither possible nor desirable to enumerate, much less describe, them here. Merely as instances I may refer to the sore fingers of girls who plait straw, the hypertrophied skin on the left hand of calico-printers, and the callosities under the tuberosities of the ischii in these same workmen. Tracy (*op. cit.*) presents many of these affections in a table, in most of which I am not able to see much that is distinctive. These callosities naturally are found mostly on the fingers and hands; but it is a refinement to attempt to point out distinctive kinds of them in most cases. It is sufficient for our purpose here merely to allude to the fact that many of the trades have been analyzed and dissected, as it were, by enthusiastic hygienists, and that in some cases the discoveries made by these scientists have been little more than callcsities on the hands and varicose veins in the legs. Borchard ("Maladies des Menuisiers et des Ebenistes," Paris, 1859), some years ago, tcok up the case of the carpenters and cabinet-makers, basing his paper on facts collected by Koblank, of Berlin; and was able to show little more that was distinctive in them than whitlows and varicosities. These latter were originally pointed out by Ramazzini in his chapter on the diseases of those who stand much at their work.

It is said that dentists sometimes suffer with an affection of the leg characterized by strain of the muscles and impeded circulation (Coles). The affection seems to have some remote affinities with the fatigue neuroses.

Ramazzini, two hundred years ago, pointed out that the eyes may suffer from overuse. This subject has already been referred to under the head of "Miner's Nystagmus." In many other circumstances, however, the eyes suffer from prolonged use in constrained positions. Jewellers and engravers especially feel these effects.

Among deformities incident to constrained attitudes or exercise may be mentioned lateral curvature of the spine, flat-foot, elevation of one shoulder above the level of the other, and bow-legs. Lateral curvature and flat-foot are more likely to be induced in young persons, the former by occupations which cause excessive over-action of the limbs of one side, especially in bearing heavy weights; the latter in positions which require long standing. Elevation of one shoulder, usually the right, is caused by heavy lifting and throwing. Bow-legs are seen not infrequently in jockeys and others whose calling obliges them to ride horses frequently and for long periods. A rather rare deformity in those who are obliged to carry heavy weights is spondylolisthesis, or excessive forward curvature of the spinal column in the lumbar region (Bradford and Lovett). Lateral curvature, according to Bradford and Lovett ("A Treatise on Orthopædic Surgery," New York, 1890) is not very common in workmen and women, for the reason that laborious occupations are not in general engaged in until an age at which the spine is equal to the task imposed upon it. It is seen in occupations requiring stooping. Clerks and blacksmiths present slight forms of it in which one shoulder is higher than the other. It is also seen in adolescents whose occupations require them to carry baskets or trays, thus twisting the spine. In short, these authors conclude, occupations which require constant one-sided attitudes, as in the clerk, blacksmith, artist, etc., may in certain individuals develop a lateral deviation of the spinal column as the natural result of this sustained position.

Dupuytren's contraction of the fingers is attributed in some cases to occupation. According to Paget the affection is common in locksmiths and wire-workers. Constantly repeated slight blows to the palm of the hand, as in these and other similar trades, are supposed to lead to it.

Straining at work leads to affections of the internal organs. Hernia is one of the most common of these. It is estimated that one-fourth of the human race are ruptured; it is of course impossible to say how many of these cases are due to occupation, but a certain proportion of them probably are. Ramazzini said that rupture occurs in public singers from the strain of using the voice. The heart and lungs suffer from strain, and emphysema and irritable heart are thus caused. The former is said to occur in professional players on wind instruments; the latter in soldiers and sailors and those who labor excessively. The Cornish miners suffer from the laborious effort of climbing the ladders in emerging from the deep mines.

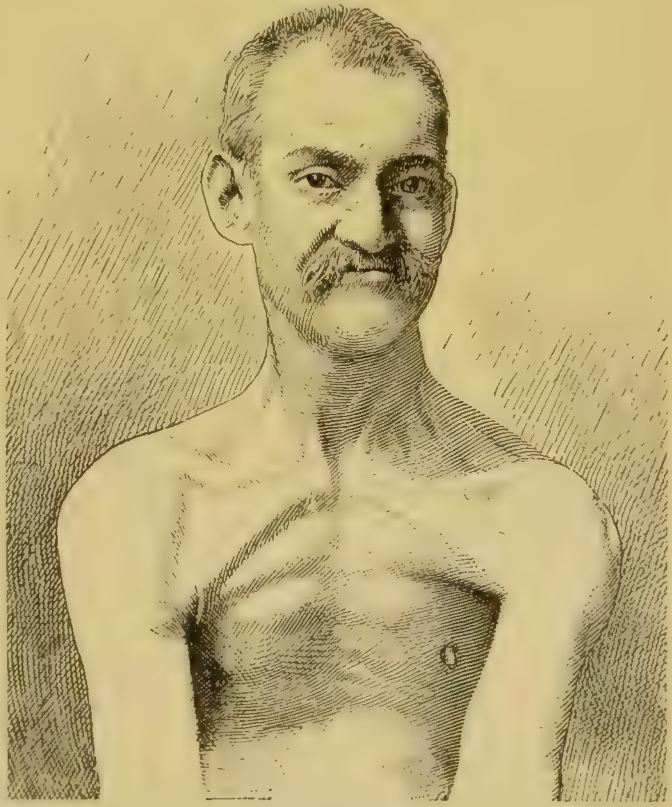

FIG. 9.—Deformity of the Chest in a Shoemaker who Worked on a Patented Machine.

In modern manufacturing processes the machine is taking the place of the hand so rapidly in many instances that the injuries and deformities caused by overuse and by constrained attitudes are being subject to radical change. Many of the old-fashioned processes which caused their own peculiar *stigmata* have disappeared or are disappearing. While therefore many of the old signs no longer exist or are no longer sure indices of occupations, on the other hand new signs are appearing. The new machinery itself not infrequently has its own way of setting its mark on men. This is a somewhat important fact to be borne in mind. It is possible indeed that powerful and complicated machinery may sometimes demand services of men which may, even short of accident, tend to mark them conspicuously and even to deform them. In illustration of this fact I append here the picture of a man (Fig. 9) who has quite a well-marked deformity and depression of the sternum, which he attributes, apparently correctly, to the action of a machine with which he worked. The man is by trade a shoemaker; in recent years he has been working with some patented machine for making shoes. This machine requires that at a certain stage the shoe be brought with some force against the sternum. The frequent repetition of this act has resulted, in a man not very robust, in a deformity of the breast-bone consisting of a depression with a rather peculiar prominence of the shoulders and clavicles.

Among the rather more conspicuous deformities due to attitude assumed in occupation may be mentioned the housemaid's knee, which is due to enlargement of the bursa of the ligament of the patella from much kneeling. A similar condition, it is said, is met with in roofers, who stoop and kneel much at their work. An exceedingly wearing trade, which may give a similar result, is that of floor-planing, which in most of our large cities is a specialty, distinct from the trade of a carpenter.

Locomotive engineers and firemen are exposed to a peculiar strain both of muscles and nerves which sooner or later is said to show its effects in many of them. This is so especially in the case of the engineers or drivers. The locomotive driver sits for long hours together in a cramped position in a small space, enduring the incessant jar of the locomotive and being under the necessity of constantly exercising alertness of mind and especially of the senses of sight and hearing. In addition he is much exposed to the inclemencies and changes of the weather. There is a popular notion, among the men themselves I believe, that the calling has a tendency to make them fat. Some scientific observers have shared this opinion. These engineers, according to Tracy, suffer in years with a diminution in the faculties of sight and hearing, and with dull pains in the lower limbs and a feeble and impaired gait. Layet says that both engineers and firemen are subject to varicocele.

POISONING.

BY

BEAUMONT SMALL,

OTTAWA.

POISONING.

VEGETABLE POISONS.

Aconite.

ACONITUM, order Ranunculaceæ, is a genus containing numerous species, nearly all of which are active poisons; a few are devoid of virulent properties. It is indigenous to the mountains of central Asia, but the different species are very widely distributed throughout the mountainous districts of Europe and America. Some species are cultivated as ornamental plants and are frequently met with in gardens. The Asiatic species, *Aconitum ferox,* Wallitch, is considered the most poisonous of all. Its deadly properties are well known to the natives who make use of the juice for the preparation of poisonous arrows.

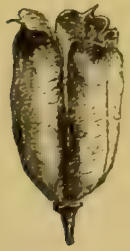

FIG. 10.—Seed-vessel of Aconitum Napellus.

Aconitum napellus, L., which is recognized as the official source of the drug by the various pharmacopœias, is the common species growing throughout Europe, particularly in the central mountainous districts. It does not occur in this country nor in Great Britain except under cultivation. The stem is simple, from one to three feet in height, bearing numerous alternate leaves, and terminating in a long, close terminal raceme. The flowers are blue or purplish in color and helmet-shaped. The seed vessel is formed of three or five earfuls, somewhat diverging, containing numerous angular, wrinkled seeds. The leaves are palmately divided into three or five wedge-shaped parts and these again deeply incised. The lower leaves are more numerous and more compound than the smaller upper leaves. The root is a simple tapering tuber giving off its rootlets profusely. In length about four inches and one inch thick at its upper portion, it somewhat resembles the parsnip and horseradish for which it has been mistaken. Each root is an annual, but

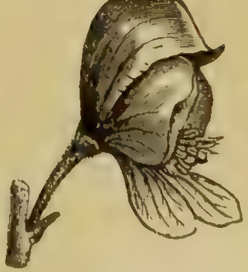

FIG. 11.—Flower of Aconitum Napellus—Monkshood.

springing from its side is a stolon terminating in a tuber which develops as the old root dies and bears the stem of the succeeding year. The leaves and root are the parts employed, the latter being considered about six times stronger than the leaves. It is directed to be gathered in the spring of the year before the leaves appear, at which period it is strongest; its activity continues until the close of inflorescence, when it becomes much weaker and gradually inert. The leaves are most active just at the beginning of inflorescence.

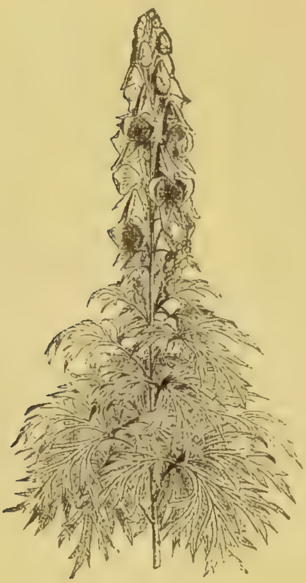

Fig. 12.—Aconitum Napellus Plant, showing alternate leaves with terminal raceme.

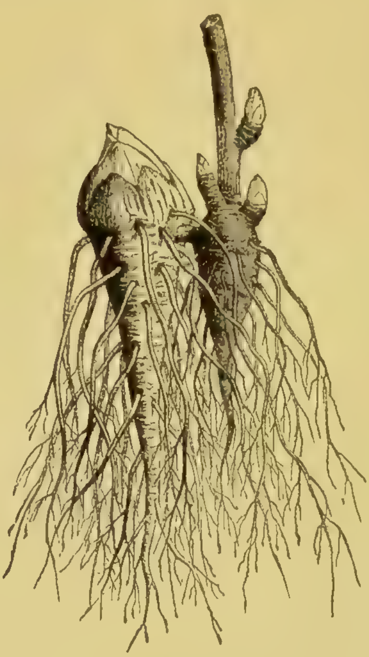

Fig. 13.—Aconite Root.

On this continent there are but two species, *A. uncinatum*, L., and *A. Fischeri*, Reich. Both of these have been very carefully studied, physiologically and botanically, by Professor Lloyd ("Drugs and Medicines of North America," 1885). The first occurs in the Allegheny Mountains from Pennsylvania southward. It was found to be almost inert and devoid of any poisonous action. The other species is of greater importance, as it contains a large percentage of aconitine and has all the properties of *A. napellus*. It grows in abundance on the Rocky Mountains and Pacific slope, throughout the United States and Canada, extending northward into Alaska. This species differs in appearance from *A. napellus*. The stem grows four, six, and sometimes ten feet high; the foliage is not so dense and the terminal raceme is loose, each flower having a stalk one or two inches in length; the flowers are blue but vary to white; the leaves are orbicular in shape and not so deeply dissected. This species varies much, approaching the *A. napellus* in some forms, and in others, the species of northern Asia.

The properties of aconite depend upon the alkaloid *aconitine* which exists in the plant in combination with aconitic acid, but our knowledge of the chemistry of the plant is still unsatisfactory. *Napelline, homo-napelline, aconine,* and other alkaloids have also been separated and described, but the distinctive properties of each are unknown; some undoubtedly are the result of the analytical process (*Journal of the Chemical Society*, 1877, 78, 91, 92). *Pseudaconitine* obtained from *A. ferox* is similar but more powerful than aconitine. In the British Pharmacopœia aconitine is described as "a white usually amorphous solid, soluble in 150 cold and 50 of boiling water, much more readily in alcohol, ether, and chloroform, alkaline in reaction, causes a tingling sensation followed by numbness when rubbed on the skin." In the United States Pharmacopœia it is not recognized.

Aconitine is of very variable strength, some samples proving almost inert. In appearance there is also a great difference. Squibb (*Ephemeris*, November, 1882) examined and described a number of samples as prepared by different makers; *Petit's*, hard, white crystals; *Morsen's*, a dirty-white powder; *Hottot's*, a white powder; *Merck's*, a yellowish powder; *Hopkin's*, a concrete gummy mass of a greenish-brown color; *Friedlander's*, a concrete grayish-white mass; *Duquesnel's*, a white crystalline form. The last-named, which is considered to be a nitrate of aconitine, is one of the most active and permanent forms. The aconitine of the British Pharmacopœia is supposed to contain a certain amount of pseudaconitine which adds to its activity.

The preparations of aconite are not numerous, but they are of importance in their relation to toxicology on account of the difference in the strengths of the official preparations in different countries. The tincture, so universally employed, shows a great diversity. That of the United States Pharmacopœia contains thirty-three per cent. of the drug, of the British Pharmacopœia sixteen per cent., of the French twenty per cent., and of the German ten per cent. There is also "Fleming's" tincture, which is still to be found in the shops, with a strength equal to twice that of the United States Pharmacopœia. It is also important to remember that the tincture of the old pharmacopœia was made from the leaves; the present pharmacopœia directs the root to be used and makes the tincture one-fourth stronger. There is also a decided difference in the extracts. The United States Pharmacopœia orders a solid extract, the dose of which is $\frac{1}{6}$ to $\frac{1}{3}$ grain, that of the British Pharmacopœia is a green extract, from the fresh leaves, the dose of which is $\frac{1}{3}$ to 1 grain. The liniment of the British Pharmacopœia is very powerful, being prepared from 1 part of the root to $1\frac{1}{2}$ of spirit; a two-per-cent. ointment of the alkaloid is also official.

A simple test, proposed by Dr. Squibb, for the estimation of the relative strength of any preparation, is to compare the effects of solutions of the drug upon the tongue and mouth. One fluidrachm is to be placed in the mouth for one minute and the rapidity of onset and duration of the tingling compared with the effects produced by a standard solution. In this way he estimated that 1 grain of powdered aconite root is equal to 1 grain of commercial aconitine, $\frac{1}{8}$ grain of Merck's aconitine, $\frac{1}{83}$ grain of Merck's pseudaconitine, $\frac{1}{121}$ grain of Duquesnel's aconitine, 1 minim of the fluid extract, 2.66 minims of the tincture, United States Pharmacopœia, 8.43 minims of the tincture, British Pharmacopœia, and 9 grains of aconite leaf.

Aconite is a powerful sensory and motor depressant, causing death by paralysis of the heart and circulation. Its earliest effects are manifested upon the peripheral terminations of the sensory nerves and then upon the spinal centres, affecting the motor nerves from within outward. The centres of the medulla are equally depressed, producing a lowering of the blood pressure and an enfeeblement of the heart, which is increased by the action of the poison upon the intrinsic ganglia. The respiratory centre is also affected and respiration becomes weakened. The brain is not influenced and consciousness remains clear until near the end. Although the motor nerves may be greatly depressed the muscular tissue remains unaffected and retains its irritability until the toxic action is far advanced. When death takes place the heart remains in diastole. Previous to death the effect of the poison upon the circulation and respiration retards the proper oxygenation of the blood and a condition of cyanosis may be added to that caused by the poison.

The *symptoms* of poisoning commence as a sense of numbness and tingling of the tongue and fauces which is very characteristic of aconite. This may extend to the lips and face, and to the œsophagus and stomach. Pain, constriction of the throat, difficulty in swallowing, nausea, and vomiting may follow. The numbness and tingling extend to the extremities and surface of the body, and muscular weakness with inability to move or walk may be very decided. This weakness also involves the muscles of the body and chest. The pulse, which at first was slow and full, becomes rapid, feeble, irregular, and intermittent. The surface becomes pallid and moist. Respiration becomes weaker and more shallow, and the bodily temperature falls. As the respiratory and circulatory organs fail, there is an increase of carbonic acid in the blood, which causes a lividity of the lips, swelling of the extremities, cold perspiration, labored breathing, and disturbances of the cerebrum, ending in convulsions and coma.

The poison is rapidly absorbed from the stomach and from the surface of the body, many instances of poisoning having arisen from external applications of the alkaloid. Excretion is slow in comparison with absorption and this allows of an accumulation of the poison in the system, as about six or eight hours elapse before its excretion is completed.

In whatever form aconite is taken its toxic action is rapidly manifested. Within five minutes the system may be under its influence, and within as many hours, if a sufficient quantity has been taken, the patient will have succumbed. When the roots or leaves have been the cause of poisoning, the onset of the symptoms and the course may be slower, death not taking place for several hours; but when the conditions for absorption of the poison are favorable, the reports of cases show that death generally follows in from two to five hours. When the amount is excessive death may follow much more rapidly. Haines ("Hamilton's System of Legal Medicine") states that it has occurred in eight minutes. When the system withstands the effects of the poison from five to six hours, every hope of recovery may be entertained. The cardiac and muscular weakness may persist for several days, also the gastric disturbance.

There is much uncertainty as to the smallest quantity that is likely to cause death, or the maximum dose that may be recovered from, owing to the varying strength of the drug and its preparations, and to the alkaloid itself. Blyth has very concisely presented and compared the investigations of Plugge (*Archives de Pharmacie*, Jan. 7th, 1892; Transactions of the International Medical Congress, 1881), and Harley ("St. Thomas' Hospital Reports," 1874), and considers that of the aconitine of the British Pharmacopœia and the French Codex $\frac{1}{32}$ grain (2 milligrammes) is the least fatal dose for an adult. This is based upon the amount required to cause death in animals and equals .28 milligramme per kilogramme of body weight. With these figures as a guide he points out that the fatal quantity is present in 30 minims of the British Pharmacopœial tincture, which contains about one per cent. of the alkaloid. Applying this to the United States Pharmacopœial tincture, which is double the strength of the other, 15 minims would represent the same amount. Although this is much below what is usually considered a dangerous dose, it becomes a serviceable guide in considering this important point. Turning to clinical evidence, it is found that very much larger doses have been taken in fatal cases, and many instances are on record where quantities, greatly in excess of what has caused death in others, have been taken without a fatal termination. The reports of such cases, however, are very meagre and incomplete, and when vomiting has occurred it is difficult to estimate the quantity that has been absorbed. Of the alkaloid, $2\frac{1}{2}$ grains were taken and the patient recovered completely in twenty-four hours (*Lancet*, 1848, I., 14). An adult was given $\frac{1}{7}$ grain of aconitine nitrate in divided doses, extending over a period of forty-eight hours; alarming symptoms arose but the patient recovered. The physician who had prescribed took what was equal to $\frac{1}{16}$ grain and died in five hours (Blyth). When the crude drug or its preparations have been taken, much larger quantities, proportionately, are required to cause poisoning, as the absorption is slower and emesis more frequent. Numerous deaths have followed the use of the leaves and root, but the quantities have been indefinite. A child, aged two years, ate of the leaves and died in twenty hours (*Lancet*, 1856, I., 715). A child, aged fourteen years, ate of the leaves and died in seven hours (*Medico-Chirurgical Review*, 1844, 256). An adult drank a decoction of leaves and stalks and died in one hour (*Medical Times*, October 18th, 1845). Blyth states that 60 grains of the root have proved fatal and estimates that ten grains would be dangerous.

Of the preparations twenty-five drops of the tincture have caused death, also four grains of the extract (Haines); two drachms of the liniment (*British Medical Journal*, 1886, II., 680); nine minims of liniment (*British Medical Journal*, 1891, II., 994). On the other hand recoveries have followed the ingestion of one drachm of the tincture (*Edinburgh Medical Journal*, August, 1870); three drachms of Fleming's tincture (*Lancet*, July, 1866); and twenty-five drops of fluid extract (*British Medical Journal*, July, 1870).

An adult woman, after her tea, took about two drachms of the tincture. In about twenty minutes she was found conscious but in a state of prostration, pulse scarcely perceptible, skin cold and clammy, eyes staring. About five hours later she made an effort to sit up in bed and refused medicine; almost immediately the pulse ceased and she fell back dead. Post-mortem fourteen hours later. Surface of body pale and bloodless, with slight discoloration about the neck and legs; the pupils were dilated. On opening the skull the vessels of the brain were not enlarged nor was the dura mater congested, the quantity of blood in the sinuses was lessened, and there was very little fluid in the ventricles. Heart healthy, both ventricles filled with dark fluid blood; lungs not congested, healthy; abdominal organs healthy. Mucous membrane of larynx redder than natural; œsophagus pale; mucous membrane of stomach paler than natural, except along the greater curvature, where some patches of congestion were found, not, however, definitely of an inflammatory character. The fluid on the lining membrane of the stomach, when brought in contact with the tongue, produced the peculiar tingling of aconite (*Lancet*, 1855, I., 467).

Child, aged 2 years, ate green leaves early in the afternoon. Was noticed to be uneasy and restless, and some hot tea was given; very soon it became suddenly ill, had pain in the stomach and throat, mucous secretion in the mouth, vomited its dinner and a quantity of leaves. Pain and tenderness in the abdomen became very severe. Five or six hours later the child became collapsed and died about eighteen hours after taking the poison. Post-mortem sixty hours after death showed stomach highly inflamed throughout; no food or traces of the leaves were present; in the intestines there were "patches of intense inflammation approaching gangrene." Bladder full of urine; heart full of fluid; blood-vessels in the same condition (*Lancet*, 1856, I., 715).

A man, 57 years old, ate aconite root at dinner in mistake for horse-radish. In about three-quarters of an hour he had severe burning and numbness of lips, mouth, and throat, which extended to the stomach, vomiting ensued and continued; the extremities were cold, head bathed in perspiration, with eyes glaring; there was marked trembling; consciousness was preserved throughout; shortly before his death he walked to the water-closet, and on his return went to bed and died in a few minutes, four hours after eating the root.

His wife also ate some and had tingling of the mouth, lips, throat, and stomach, with violent vomiting; the hands, arms, and legs were benumbed, and she was unable to stand; articulated indistinctly; there was constriction of the throat; sensibility greatly impaired; face and throat almost insensible to the touch; body and extremities cold. In five or six hours she began to recover (Pareira, Bently and Redwood, 1,001).

A man, aged 38 years, drank an infusion of aconite and burdock roots, estimated equal to one grain of aconitine. Was found in a state "akin to catalepsy"; the body and extremities were cold, and rigid, and the man was totally unable to move or speak; respiration scarcely perceptible; no pulsation at the wrist or in the axilla; insensible to prick of pin, etc. Three hours later he was able to move the eyes and mumbled a little; pulse 70, temperature 97°. Six or seven hours later was able to converse; whenever there was sensibility he complained of intense tingling and numbness. For twenty-four hours he remained paralyzed in the lower extremities, and for some days there was weakness. Patient stated that he had been conscious from the first and was aware of his surroundings and what was done. Treatment consisted of apomorphine, croton oil, draughts of coffee, and the "usual antidotes" (Detroit Emergency Hospital Reports, September, 1892).

Adult, male, chewed "a small quantity" of aconite root. This was followed immediately by tingling of tongue and fauces; numbness of face; vomiting; he became very restless; the burning in the stomach became severe, and the tingling extended to all parts of the body. He gradually lost the power of movement in the limbs; face became anxious; eyes suffused, pupils moderately dilated; respiration labored, 64 to the minute; pulse 66, small and feeble. About four hours after taking the root the man's condition became much worse. The tongue became red and swollen; the pulse intermittent, feeble, and slower. The tingling and numbness increased. The patient began to improve after nine hours, consciousness having remained clear throughout (Blyth, "Poisons," p. 326).

Adult, female, after breakfast swallowed about three drachms of Fleming's tincture. In half an hour mouth and throat were hot and tingling with a sensation of choking; arms and legs became benumbed; a quantity of milk was taken and patient walked about one-half mile. In two hours, when seen by a physician, she had pinched features, skin cold and clammy, breathing hurried and short, pulse 45 and very feeble, with paroxysms of choking. The stomach was evacuated, heat applied to the surface, and stimulants given. The following morning the woman was comfortable but weak, with a slight burning and tenderness at the epigastrium (*Lancet*, 1866, II., 34).

A man drank about a tablespoonful of Fleming's tincture. Almost immediately there was burning of the throat, mouth, and lips extending to the stomach; half an hour later there was numbness in the extremities, which became cold, with a sense of weight and inability to lift the feet from the ground; the breathing was hurried and labored; consciousness remained undisturbed. About five hours later the face was flushed; conjunctivæ injected; extremities cold and clammy; respirations 36; pulse small and soft, 80; mouth frothy with saliva. The case was treated with emetics, stimulants, and heat. The following day the man complained of headache, feebleness, and cramps in the muscles of the legs, which lasted for several days (*Lancet*, 1867, I., 238).

An adult took a pill of one-sixth grain crystallized aconitine. Almost immediately emetics and the stomach-pump were used. In forty minutes the face was pale; voice almost lost; there was a feeling of impending death followed by loss of consciousness. The pulse was small and rapid; respirations feeble and irregular; extremities cold; skin clammy. The treatment consisted in the application of heat to the surface, and the administration of caffeine 15 grains, ether 45 minims. Two hours later the pupils dilated; the pulse was almost lost, 30 per minute, and respirations were equally weak; there was muscular trembling. Caffeine 15 grains and ether again used. Symptoms began to improve and the danger was over in nine hours. The patient did not fully recover for a week (*Therapeutic Gazette*, 1894, 120).

In the *treatment* of these cases the primary object is to remove from the stomach, by emesis as well as by the aid of the stomach-pump, such portions of the poison as remain unabsorbed. At the same time solutions of tannic acid, or iodine in solution with iodide of potassium, may be administered for the purpose of retarding the absorption of the active principle. Heat to the extremities and surface of the body is called for, also stimulants to support the enfeebled heart. Throughout the continuance of the symptoms rest in the recumbent position should be maintained, and any sudden effort or attempt to rise should be avoided on account of the danger of fatal syncope. The bladder should be catheterized, as in the early stages the action of the kidneys is increased and much of the poison is excreted by this channel.

The recommendation of alleged physiological antidotes to aconite is based upon theoretical considerations without much clinical evidence to support them. Those that are proposed are strychnine, atropine, caffeine, and digitalin. They all exert a stimulating action upon the heart and respiration, and when administered subcutaneously are rapid in producing these effects. They must, however, be used with a certain amount of caution, as in excessive doses they also act as depressants upon the heart.

The *post-mortem appearances* are not very definite; a reddening of the mucous coats of the stomach may be present, and in some cases there have been found local areas of hyperæmia and congestion. The vessels and organs present the conditions which would naturally be looked for, given the cyanotic state of the blood. The arteries and veins are full of dark blood, the heart is dilated, and all the organs are congested. The pupils are dilated. Traces of the poison may be present in the stomach and fæces. Experimentally the alkaloid has been detected in the urine, blood, and various organs, but in cases of poisoning the quantity absorbed is so small that it readily escapes detection.

Belladonna.

Deadly nightshade, *Atropa belladonna*, Linnée, belongs to the well-known order of Solanaceæ, all the species of which possess poisonous properties to a greater or less extent. Other well-known genera of this order are solanum, hyoscyamus, datura, physalis, nicotiana, capsicum, and mandragora.

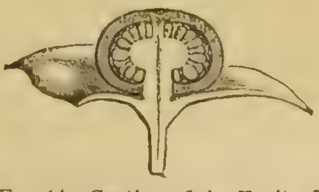

Fig. 14.—Section of the Fruit of Atropa Belladonna, showing seeds.

Belladonna is a perennial herb growing from three to five feet in height. The stem is erect and smooth, terminating in a number of branches which are slightly downy. The leaves are alternate but situated in pairs of unequal size; they are shortly petioled, broadly-ovate, acute, entire, smooth; the larger leaves 6 to 8 inches in length. The flowers are solitary and drooping, of a dull purple color, about an inch long. The calyx is five-parted, remaining upon the fruit. The corolla is bell-shaped. Fruit is a violet-black, shining berry, the size of a small cherry, two-celled, containing numerous seeds. The whole plant when bruised emits a fetid odor.

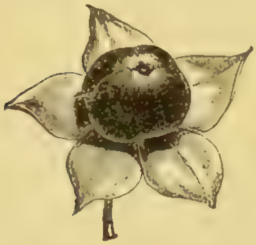

FIG. 15.—Berry of Atropa Belladonna; about two-thirds of the natural size.

Belladonna is indigenous to central and southern Europe and western Asia; it is found in Germany, Switzerland, France, Spain, Italy, Hungary, and extends into Asia. It does not occur north of the Baltic Sea. In Great Britain it is extensively cultivated; in many localities it has escaped from cultivation and is found growing wild. It has not been introduced into America and is only occasionally met with under cultivation. In this country the name is frequently given to the different species of solanum, or bittersweet, which are not uncommon plants.

All parts of the plant are poisonous and the root, leaves, berries, and seeds have been the cause of accidents; the attractiveness of the flowering plants and the sweet taste of the berries being particularly seductive. The roots and leaves are official in the United States and in Great Britain, in France the seeds also are official, in Germany the leaves only are recognized. All the preparations of belladonna are concentrated and powerful. The *linimentum* is an alcoholic extract of the root with a small percentage of camphor, the *emplastrum* contains twenty per cent. of extract, and the *unguentum* ten per cent. The analogous preparations of the United States Pharmacopœia and British Pharmacopœia do not vary much in strength, although the methods of obtaining them vary somewhat. A source of danger, however, might arise in the solid extracts, the *extractum belladonnæ foliorum alcoholicum*, and the *extractum belladonnæ alcoholicum*, being administered in doses of less than one-half grain, while the *extractum belladonnæ*, an extract made from the green leaves, is several times weaker than the others and given in doses ranging from one to three grains.

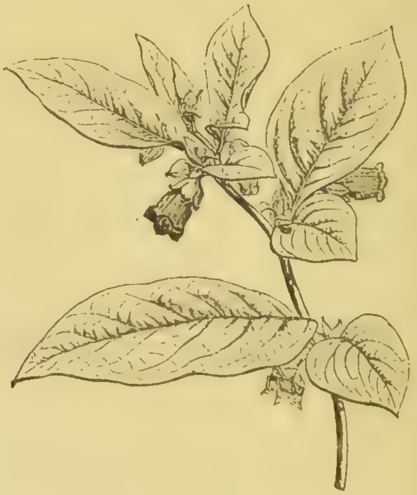

FIG. 16.—Flowering Branch of Atropa Belladonna.

The properties of belladonna depend upon the alkaloid *atropine* which is present in all parts of the plant to the extent of one-fifth to three-fourths of one per cent. A second alkaloid, *belladonnine*, is also present, but its influence upon the physiological action of the plant is of little value, as it is of uncertain composition and exists in very small amounts. The sulphate of atropine is the official salt.

The toxic action of belladonna is that of a vasculo-cardiac poison, death following a paralysis of the vaso-motor and cardiac centres.

When administered in therapeutic doses, the early acceleration of the heart arises from the depressant effect of the drug upon the inhibitory nerves which is coincident with the general stimulation of the medulla oblongata. In fuller doses this depressant action extends to the ganglia of the medulla, with a consequent lowering of the blood-pressure and enfeeblement of the heart. The effect upon the respiratory organs is similar but less marked. There is a depressant action upon the peripheral terminations of the pneumogastric and an early stimulation of the respiratory centre followed by a depression. The respiration, however, is not so markedly influenced as the pulse and in a great number of cases the disturbance is not very evident. Upon the brain it produces a restless intoxication, occasioning an active delirium which gradually passes into stupor. If the effect is more profound the period of excitement may be rapidly passed and replaced by a state of coma. The effect upon the spinal cord is the same as upon the higher centres—a short period of excitation followed by depression.

One of the earliest evidences of the paralyzing action of belladonna is its well-known effect upon the secretory organs, particularly the salivary and sudoriparous glands, the checking of the secretions arising from the depression of the terminal nerve fibres. The dilatation of the pupil is also the result of a loss of tone of the constrictors of the iris.

The *symptoms* of poisoning are generally very distinct. The pupils are dilated, the skin is dry and hot, the mouth and throat are parched, and dysphagia is present. There may be restlessness and delirium, but coma is more commonly met with; convulsions also may occur but they are not frequent. The respiration may not be affected, or, in severe cases, it may be rapid, shallow, and stertorous. The pulse is, however, always increased in frequency and is weakened in proportion to the severity of the poisoning; the blood pressure is equally lowered. A very characteristic sign is the erythematous rash that arises from the vaso-motor paralysis. It begins early in the course of the symptoms and is very persistent. With the rash an elevation of temperature takes place. The muscular system becomes weakened; numbness of the surface, incoördination, inability to walk, and paralysis may be present. The loss of sensitiveness in the mucous coat of the bladder causes a retention of urine; the secretion of the kidneys is scanty and tenesmus and dysuria may be present. When the effects of the poison are severe and prolonged, the inactivity of the heart and lungs causes an insufficiency of oxygen in the blood, and all the symptoms of cyanosis are added to those peculiar to the poison itself. It is this condition of asphyxia that is usually the immediate cause of death.

The absorption of the alkaloid is rapid, and the interval between the ingestion of the poison and the onset of the symptoms is very brief. In experimental work, it is repeatedly stated that its influence upon the pupil begins before the hypodermic injection of the drug is completed. When the poison has been taken by the mouth the earliest symptoms are its local action upon the mouth and fauces and the dilatation of the pupils; in some instances, where the alkaloid is the form of the poison or where its absorption is free, the cerebral symptoms begin almost immediately, and the earliest evidence of the poisoning will be a restless intoxication or a state of stupor simulating apoplexy. In many cases of poisoning with suicidal intent the patient will be found to have indulged too freely in alcoholic drink, and is possibly even verging on delirium tremens. Under such conditions there may be difficulty in recognizing the signs of poisoning, or the delirium may simulate alcoholism and the true cause be overlooked.

The course of the attack is also rapid. Within a few minutes after the absorption of the poison excretion commences, and the alkaloid may be detected in the urine. In about five or six hours, if the function of the kidney is not checked, the greater part of the poison will be removed from the system, and within ten or twelve hours it will generally be found that the urine is free from any traces of it. Corresponding to the period of excretion, it will be found that an improvement in the condition of the patient generally begins in about four or five hours. If the patient survives that time without signs of impending death, or without an increase in the severity of the symptoms, a favorable prognosis may be given and a rapid improvement looked for. The earliest signs of recovery are a slowing of the pulse, a fading of the rash, and glimpses of returning consciousness. Once turn for the better shows itself, the danger is rapidly overcome, but the system remains under the influence of the drug for several days. The cerebral symptoms may persist, the pupils continue dilated, the throat remain dry, or the paretic condition persist. The bladder may also continue inactive.

When the form of the poison does not allow of a rapid absorption the onset may be delayed and the course greatly prolonged. Instances are recorded where no effects arose for five hours. When a fatal termination is the result of the poisoning, death usually takes place in a few hours. It may occur much more rapidly. An instance is recorded in which death in five minutes followed an hypodermic injection of the alkaloid.

The smallest quantity of the alkaloid that is likely to prove fatal is very uncertain, but authorities are quite generally agreed that one grain will probably be a fatal dose. Blyth states that $\frac{1}{30}$ grain hypodermically and $\frac{1}{12}$ grain by the stomach has caused death. Of the preparations, doses of 1 drachm of liniment, 2 drachms of solid extract, or 1 drachm of tincture (*Lancet*, February, 1867) have proved fatal. The external application of an ointment containing 3 grains of the sulphate of atropine and 2 drachms of lard to a raw surface caused death in two hours (*Annales de Thérapeutique*, 1867, 9). On the other hand very large doses have been recovered from. One ounce of liniment, 3 drachms of Fleming's tincture (*Lancet*, July, 1866), 1 ounce Fleming's tincture (*British Medical Journal*, December, 1872), and in a child 3½ grains of the alkaloid (*British Medical Journal*, 1889, 1, 1051) were taken without causing death. Three of the fresh berries have caused death (Ziemssen), and thirty have been recovered from (Taylor).

These varying results are to be explained in several ways. The production of emesis or removal of the poison with the aid of the stomach-pump prevents the absorption of much of the poison. The presence of food in the stomach also delays the action of the poison and facilitates its expulsion. The unreliability of the crude drug and of its preparations is also an important factor.

Boy, aged 16 years, took 2 drachms of solid extract: in ten minutes he became convulsed and insensible, eyelids closed, jaws set; convulsions and prostration increased, and death followed in three and a half hours (*Lancet*, 1869, II., 327).

Woman, 66 years old, drank about one teaspoonful of the liniment at 11 A.M.; was seen at 2 P.M., delirious, pupils dilated; pulse 126. Stomach-pump was used and opium given; the delirium subsided, and the woman became comatose. At 6:15, the pulse was 100, respiration not noticeable; artificial respiration was persisted in for some time. Death took place sixteen hours after the poison had been swallowed. Forty minims of tincture of opium and $\frac{11}{12}$ grain of morphine acetate were given during that period (*Lancet*, 1870, II., 83).

Man, 39 years old. One ounce of the liniment was swallowed at 5 A.M.; he remained conscious twenty minutes, then became delirious; pupils dilated, twitching of muscles, skin dry and anæsthetic. At

10:30 the skin began to act and the patient urinated; delirium subsided, but he remained unconscious until 4:30 next morning; during latter half of the period of unconsciousness the patient kept going through an automatic movement as if taking snuff. The treatment consisted in the administration of emetics, cathartics, hypodermics of morphine, and heat to the extremities and hot sponging (*Lancet*, 1884, I., 370).

Boy, aged 7. Took $\frac{5}{6}$ grain atropine. "In a few moments" became wildly delirious, and continued so for three days; on the fourth day he fell asleep and awoke recovered. Treatment consisted only in the administration of 15 grains of tannin.

Child, 3 years old, took an unknown amount of atropine at 9 A.M.; at 12 was restless, wildly crying, with twitching of extremities, pupils dilated, red skin: The stomach was washed out; $\frac{1}{20}$ grain morphine hydrochlorate was given and followed in fifteen minutes by $\frac{1}{12}$ grain which caused sleep, and the child gradually improved (*Therapeutic Gazette*, 1894, 383).

Woman, 35; took 3 drachms of linimentum before breakfast. "In a few moments" complained of her eyes and soon fell down "in a fainting fit." Face suffused, vessels throbbing, pupils dilated; pulse 160, respiration rapid, soon became slower, weaker, and at times almost ceased. In about eight hours the woman became conscious, passed urine, and improved rapidly. No delirium, no rash. Treated by washing stomach, $\frac{1}{2}$ grain morphine, hypodermic of brandy and ether and $\frac{1}{30}$ grain strychnine nitrate (*Montreal Medical Journal*, XXII., 686).

A woman, æt. 21, had taken 4 grains of atropine sulphate when intoxicated. In twelve hours the face was flushed, lips, tongue, and mucous membranes dry and parched, pupils dilated, conjunctivæ injected; there were twitchings of the fingers and toes with tremor; the woman was delirious, constantly moving in bed and picking the bed clothes, muttering, and pointing to imaginary objects. Temperature 101° F., pulse 135, respirations 32. No vomiting; retention of urine, nearly 40 ounces being drawn off. One ounce of castor oil with 8 minims of tincture of opium were ordered. The patient gradually improved. Three minims of a hypodermic solution of morphine (ten per cent.) were given and repeated twice. The following day she was conscious, with pupils dilated, skin dry, temperature 103°, pulse 128. The symptom of dilated pupils remained for one week with dry skin and constipation (*Lancet*, November 16th, 1889).

Treatment.—This should be pursued with the expectation of success, as the majority of cases tend to recovery. The first indication is to remove from the stomach such portions of the poison as may remain unabsorbed. Vomiting rarely takes place in belladonna-poisoning and it is only with difficulty that the stomach is made to respond to emetics. If the stomach-pump is available it may be used much more effectively in thoroughly cleansing the stomach. The use of charcoal or tannic acid may be combined with this procedure for the purpose of rendering the alkaloid less soluble and retarding its absorption.

As physiological antidotes, veratrine, physostigmine, hydrocyanic acid, and many other drugs have been proposed upon theoretical grounds, but they are rarely or never employed at the present day, as more effective and less dangerous remedies have been found in morphine and pilocarpine.

Opium has for many years been employed as a physiological antidote on account of its alleged antagonistic action upon the heart and respiration. Experimental work, however, has never supported this view. During recent years attention has been redirected to the subject and the actions of opium and belladonna upon the circulatory and respiratory organs have been carefully studied. The result is that not only have the investigators failed to discover these antagonistic properties, but it has been shown that with both drugs the ultimate effects are to paralyze these organs. Very many instances are recorded of the favorable action of opium in cases of belladonna poisoning, but how these beneficial effects are to be accounted for is undetermined. It is of undoubted service in the early stages to relieve the distressing throat symptoms and to overcome the restlessness and delirium. The quiet sleep that follows its use proves of decided value and from it the patient awakens much improved. In advanced stages, however, or where large quantities of the poison have been absorbed, opium has never been shown to be of much value and the wisdom of its employment is questionable. The narcotic action of opium is known to be increased by belladonna, and this, taken with its unfavorable action in overdoses, would indicate that the antidote must be used with caution when the coma is profound and circulatory failure and collapse are imminent. When employed the morphine should be administered subcutaneously, in doses of one-fourth to one-half grain and repeated at intervals, the dose and frequency depending upon the severity of the symptoms and the amount of poison. The pupils may be contracted by the antidote and again dilate as its influence passes away, but the action upon the pupil is uncertain and is not a satisfactory guide.

Pilocarpine is now considered the most efficient and desirable antagonist to the poisonous action of belladonna. The action of the two upon the heart has been demonstrated to be directly opposite (*Journal of Anatomy and Physiology*, X., 187), pilocarpine restoring the inhibitory action of the vagus after it has been destroyed by atropine. It also promotes the action of the sudoriparous and salivary glands that have been checked by belladonna. Its action upon the emunctories is also supposed to favor the excretion of the poison. It has been used with varying results (*Lancet*, I., 1876, 346; *British Medical Journal*, 1, 1881, 300 and 594; *Therapeutic Gazette*, 1892, 330).

In some instances it has failed to promote perspiration or salivation, in others it has rendered the skin moist, relieved the dryness of the mouth and throat, and overcome the strangury and retention of urine. It also, as well as opium, must be used with care, and only in selected cases, on account of its depressing action upon the heart and circulation. From one-fifth to one-half grain of the nitrate of pilocarpine may be given and repeated every fifteen or twenty minutes until an impression is made. Its effect will generally continue for five or six hours.

In addition to the employment of these antidotes, ordinary measures must be adopted to overcome the cyanotic state of the blood and stimulate circulation. Artificial respiration, inhalation of oxygen, cold affusions, and warmth to the extremities are indicated. Strychnine has proved serviceable as a respiratory stimulant. Alcoholic stimulants also may be employed to avert the stage of collapse. The elimination of the poison may be assisted by having the patient drink freely of water and using the catheter if the urine is not voided. The bowels should also be emptied by purgatives and enemata.

There is little or no evidence to be obtained from the post-mortem appearances to indicate poisoning by belladonna. If the berries, or solid preparations have been taken, traces may be present, or they may have produced localized areas of redness and congestion. A redness of the mucous membrane of the mouth and throat may be present, and the dryness of the mouth and skin may be detected. The pupils may also remain dilated. The condition of the organs is that always found in connection with death from heart and circulatory failures. The alkaloid when absorbed is conveyed to all the tissues of the body and is excreted unchanged with the urine. It is also excreted with the bile, passing into the small intestine where it is supposed to be reabsorbed, as no traces are to be found in the fæces. Atropine remains in the body for a long period without decomposition. It is not destroyed by the process of decomposition and has been detected in putrid tissues.

Calabar Bean.

Physostigma, the Calabar bean, was brought under notice on account of its employment as the ordeal-bean of the natives of Calabar on the west coast of Africa. By certain tribes it was used to determine the guilt of those charged with crime. From one to twenty-five beans were administered and, if a fatal result followed, the victim was adjudged guilty; but if he escaped death, his character was considered free from stain. The bean was studied and its botanical character determined by Professor Balfour in 1859 and its physiological action was fully described by Professor Fraser in 1863 (*Edinburgh Medical Journal*, July, 1863).

It is the fruit of *Physostigma venenosum*, Balf., order Leguminosæ, which is supposed to be the only species of its genus, and none of the allied genera resembles it in its toxic action. The plant is a large climbing vine, perennial. The stem is woody below and several inches in circumference. The leaves and flowers resemble the ordinary bean-vines, the former being trifoliate and the latter in loose hanging racemes of a purplish-pink color and very showy. The pods are green and curved when young, becoming straight and brown when ripe, and contain two to four seeds. The bean is about $1\frac{1}{2}$ inches long, $\frac{3}{4}$ inch wide, and $\frac{1}{2}$ inch thick, of a chocolate-brown color, reniform in shape, with a furrow along the convex side. The testa is rough, hard, and brittle. The kernel consists of two hard white cotyledons, inodorous and without any acrid or bitter taste. The properties of the bean are confined to the kernel and are imparted in a moderate degree to water and more freely to spirits.

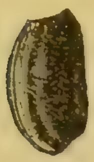

FIG. 17.—Calabar Bean; two-fifths natural size.

The active principle of the plant is the alkaloid *physostigmine* or *eserine*. It is directed to be obtained by "dissolving the (alcoholic) extract in water, adding bicarbonate of sodium, shaking the mixture with ether, and evaporating the ethereal liquid." The alkaloid is official in the British Pharmacopœia. In the United States Pharmacopœia the salicylate and sulphate are official. The alkaloid occurs as pinkish crystals slightly soluble in water and very readily in alcohol. It is altered by exposure, losing its crystalline form and becoming an amorphous mass. The salts are a fine crystalline powder, colorless, but on exposure to light becoming pinkish, and by the absorption of moisture they also become amorphous. The salts have a bitter taste and are freely soluble in water. The preparations of the United States Pharmacopœia are an alcoholic extract of the strength of one part in two, and a fifteen-per-cent. tincture. The British Pharmacopœia orders the extract only.

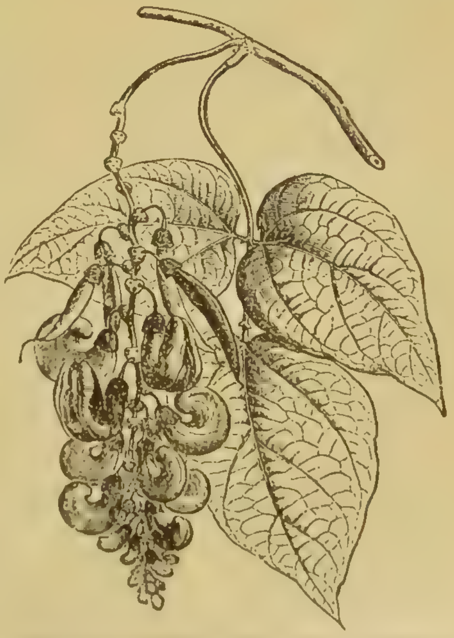

FIG. 18.—Calabar Bean Vine; flowering branch; reduced.

Symptoms.—The action of physostigmine is directed to the anterior cornua of the spinal cord, producing, after a brief stimulation, a motor paralysis and a loss of reflex irritability. It also depresses the vital centres. The respiration is weakened and in fatal cases death takes place from paralysis of the respiration. The heart's action is also depressed. The intellect is not affected, and sensation is but little influenced, unless the drug be taken in excessive doses when it also depresses the posterior cornua and lessens sensibility. The pupil is contracted by its influence through the system, and by its local action. The effect upon the stomach is not very decided; salivation, vomiting, and diarrhœa may occur but are not always present. The alkaloid is excreted by the kidneys, it has also been detected in the saliva, bile, liver, stomach, and small intestines. In the early stages there may be tetanic spasms which are due to *calabarine*, a second alkaloid which is usually present in very small quantities. The effects of physostigmine resemble poisoning by curare, nicotine, conium, or other motor depressants, and may be difficult to distinguish without the history of the case. The contracted pupil is, however, very characteristic.

The instances of poisoning are not very numerous, but some remarkable cases have been reported. In Liverpool (*Medical Times and Gazette*, 1864, 406) forty-six children once ate of the bean, all of whom recovered but one, who had taken six beans. Within half an hour there followed nausea and vomiting, with heat and pain in the stomach. Diarrhœa occurred in fifteen. In an hour the nervous symptoms began with muscular weakness, cold perspiration, shallow respirations and feeble pulse, all of which lasted for thirty-six hours.

Two children, aged six and three years, ate the kernel of one nut (*Edinburgh Monthly Journal*, 1860, 193). In forty minutes they complained of nausea and weakness. One child held his head drooping and his hands were powerless. He staggered and was scarcely able to walk. He complained of severe pain in the stomach and made ineffectual attempts to vomit. Milk was given and caused emesis. The child became quite prostrated; the pulse was feeble and slow, the pupils slightly contracted. The other had the pain in the abdomen and was listless and depressed. There was some vomiting, and purging was free. He could neither stand nor walk. His face was pale; the pupils natural. Both children had fully recovered by the third day.

Dr. Fraser, in experimenting upon himself, felt heat at the epigastrium accompanied by eructations and salivation within five minutes after taking the poison. These symptoms were followed by weakness of the extremities, giddiness, difficulty in breathing, and feeble pulse. The local application of the alkaloid will give rise to the physiological action of the drug, but no instances of fatal results from the alkaloid have been reported.

The *treatment* consists in administering tannic acid and astringents and in emptying and washing the stomach. Artificial respiration has maintained life in experiments upon animals and should be adopted to support the failing respiratory efforts. Atropine and strychnine have proved valuable as antagonists in experimental work, but have not been tested in any cases of poisoning.

Cocaine.

The leaves of *Erythroxylon coca*, Lamarck, and its alkaloid *cocaine*, which are now used so freely and with so little discrimination, have proven themselves to be very powerful drugs, and numerous accidents have arisen from their employment. The toxic effects generally occur from the use of the alkaloid, but the leaves and their preparation may also cause the same unfavorable symptoms. Prof. Germain Sée, in 1892, collected 260 instances of poisoning with 21 deaths, and since that date many more have taken place.

The fresh leaves are well known for the property they have of stimulating and sustaining the vital forces during laborious undertakings, and the deleterious effects of excessive doses are equally well recognized. They then give rise to a form of intoxication which is marked by great exuberance of spirits, talkativeness, hallucinations, restlessness, rapid pulse with quickened and spasmodic breathing, which are soon followed by depression and prostration. The same results follow its continued use, a condition of chronic poisoning being established.

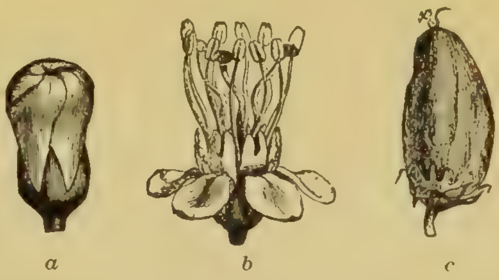

FIG. 19.—Erythroxylon Coca—*a*, bud; *b*, flower; *c*, fruit. Enlarged.

The action of coca is due to its alkaloid, which has been shown to be a protoplasmic poison. Following its stimulant effect, there is a period of over-stimulation, seen in the convulsive seizures of a tetanic character, which is more or less general and causes death by tetanus of the respiratory muscles. If this is survived an exhaustion follows which ends in paralysis of the entire nervous system and death from respiratory or cardiac failure. When the dose is large the period of stimulation may be transient, and the exhaustion and collapse appear at once. In addition to its action upon the nervous system, its effect upon the heart and blood-vessels is of importance. Acting through the nerve supply, it produces a contraction of their muscular fibres, which lessens the calibre of the vessels and increases the force of the contraction of the heart, which becomes spasmodic in character and appears to remain in a condition of systole for a short period every few beats. When the stage of collapse appears, the vessels become relaxed, the blood pressure falls, and the force of the heart diminishes. The leucocytes also are altered under the influence of cocaine, becoming spherical and rigid; they increase in size, and as the capillaries contract they are thought by some to be the cause of thromboses and embolisms by which many of the severe symptoms are explained.

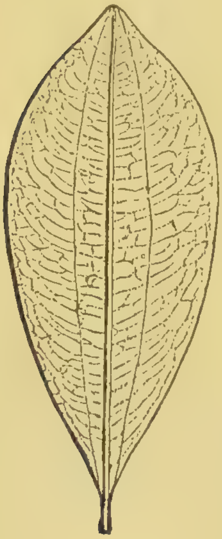

FIG. 20.—Leaf of Erythroxylon Coca, natural size; showing venation and longitudinal creases.

The *symptoms* of poisoning vary greatly, depending upon the quantity of the poison that has been taken and the rapidity of its absorption. In many cases the primary stimulating effect is very marked, but in the greater number the earliest symptoms are those of collapse and threatened death. There may be a short period of restlessness and excitement with slight cerebral disturbance, the pulse becoming rapid and hard, the action of the heart labored, and the respiration hurried, with a sense of tightness and constriction of the chest. These symptoms are more or less quickly replaced by muscular weakness, pallor, cold perspiration, feeble heart and respiration, vertigo, dilated pupils, and ultimately loss of consciousness, convulsions, and collapse. Death may follow rapidly from syncope or may be more protracted and preceded by symptoms of asphyxia. Loss of common sensibility is frequent, the extremities or localized areas of them becoming anæsthetic. There may also be nausea and vomiting, but this is not generally present.

Cocaine is rapidly absorbed when introduced into the stomach, as well as when applied to any part of the mucous tract.

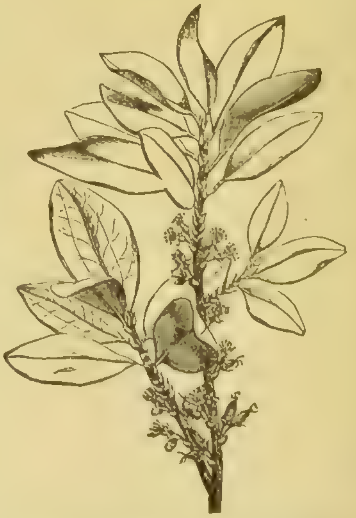

FIG. 21.—Flowering Branch of Erythroxylon Coca. One-third natural size.

The onset of symptoms may be almost immediate or may be delayed for half an hour, about ten minutes being the average. The effects of the poison have also appeared upon loosening the ligature and allowing a free circulation after an operation upon a finger or extremity in which the flow of blood had been checked. In mild cases the toxic effects pass away within half an hour, in more severe cases evidences of nervous prostration and irritability may remain for days. The action is more rapid when the solution is concentrated, and when the absorbing surface is rich in blood-vessels. The action is also supposed to be more severe when the alkaloid is absorbed into the vessels of the head or neck, and the sitting posture has been thought to exercise an unfavorable influence, the more anæmic state of the brain increasing the action of the drug. Dr. P. Mannheim in 1891 collected ninety-one cases of poisoning that had occurred up to that period (*Cleveland Medical Gazette*, September, 1891). He found that weak and nervous people are not more susceptible, nor has sex any influence. No instances of a personal idiosyncrasy were observed, as in all the fatal cases concentrated and excessive doses, too rapid injections, or intravenous injections explained the cause of death. In five of the nine fatal cases the quantity of cocaine was above fifteen grains. In all diseased conditions in which the nervous system is weakened, or where there is degeneration of the walls of the heart or vessels, it should be employed with caution.

The fatal quantity is uncertain. From experiments upon animals it has been estimated that about two grains should prove fatal (*Archiv für Experimentelle Pathologie und Pharmakologie*, XXIII., 1887), and of eleven fatal cases death in two was due to 1½ grains (*Therapeutic Gazette*, 1893, 200). The United States Dispensatory, 1890, refers to a case in which twelve drops of a four-per-cent. solution, subcutaneously, caused the death of an eleven-year-old girl in forty seconds. On the other hand very large doses have been recovered from. But it is advised that not more than three-fourths of a grain should be given as the initial dose, nor more than that quantity applied in solution; for hypodermic use not more than half a grain should be given.

"Two soupspoonfuls" of a two-per-cent. solution were injected into the scrotum for an operation on a hydrocele. After the operation the patient became faint; in twenty minutes he was in complete coma, the face and limbs moving convulsively and the pupils being dilated. He was then seized with a tetanic spasm and finally died of cardiac syncope (*Therapeutic Gazette*, 1893, 200).

A man received an injection of 20 minims of a six-per-cent. solution along the matrix after a ligature had been applied to the base of the toe. In fifteen minutes the operation was completed and the ligature was removed. In a few minutes the man became faint, the pupils were dilated, the countenance pale, respirations increased in frequency, pulse thready and irregular, registering 160 beats to the minute. Ammonia and whiskey were given, as well as digitalis and hypodermics of $\frac{1}{30}$ grain of strychnine sulphate. Later $\frac{1}{100}$ grain of nitroglycerin was injected, which improved the condition of the patient. Consciousness was not lost. He described the sensation as of a numbness ascending from the feet to the brain, calling for a constant effort to prevent unconsciousness, with great oppression in respiration, and desire for fresh air. The immediate effects lasted one hour, and the patient remained prostrated for twelve hours, suffering also from insomnia. The amount of cocaine injected was about $1\frac{1}{5}$ grains (*Therapeutic Gazette*, 1895, 11).

Treatment in mild cases consists simply in placing the patient on the back, and administering such stimulants as coffee or alcohol, and at the same time applying heat to the extremities. Amyl nitrite or nitroglycerin is recommended for its relaxing effects upon the arterioles and as a means of overcoming the anæmia of the brain. In severe cases chloroform and chloral are to be used in the early stage to remove the tetanic spasms and prevent respiratory tetanus, and strychnine or digitalis to counteract the later stage of collapse and paralysis of respiration. Morphine has been suggested as an antidote, as morphinomaniacs exhibit a great tolerance for cocaine. To be of service it should be given in full doses, $\frac{1}{2}$ to $\frac{2}{3}$ grain, immediately after the poison has been taken. When there is failure of respiration and threatened asphyxia, artificial respiration must be enforced. Should there be any probability of the poison remaining in the stomach when the patient is first seen, vomiting should be obtained and the stomach should be thoroughly washed out with the aid of the stomach-pump.

Cocculus Indicus.

Cocculus indicus is the dried fruit of *Anamirta paniculata*, Coleb., *Menispermum cocculus*, L., order Menispermaceæ, a large woody vine growing in southern Asia, India, Ceylon, and adjacent islands. The fruit has long been an article of commerce, its sole use being the illegal one of adulterating ales; it is also employed for the purpose of capturing fish by rendering the water of fish-ponds poisonous. The drug has latterly become of more importance as the source of picrotoxin. The fruit is about the size of a large pea, somewhat kidney-shaped. It consists of a large ligneous shell, covered with a shrivelled purplish-brown skin, containing a yellow-

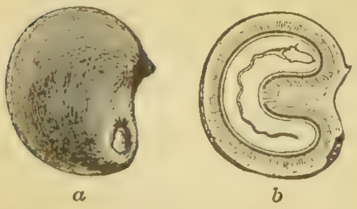

FIG. 22.—Fruit of Cocculus Indicus—*a*, entire; *b*, in section showing a slender curved embryo.

ish-white seed with an intensely bitter taste. The active principle is *picrotoxin*, a colorless, crystalline neutral substance, soluble in 335 parts of cold and 35 parts of boiling water; is soluble in 13 parts of alcohol. The presence of picrotoxin may be determined by the following method: "The substance suspected, having been brought to the consistence of paste, is to be digested with alcohol and a little tartaric acid, the liquid separated, the alcohol evaporated, the residue diluted with a little water and then treated with ether, and finally, the ethereal solution submitted to evaporation in a watch-glass. Picrotoxin is deposited and recognized by its feathery crystals and bitter taste" (United States Dispensatory, 1890). The following official test is given: "On diluting 2 c.c. of alkaline cupric tartrate volumetric solution with 10 c.c. of water, and adding a small portion of picrotoxin, red cuprous oxide will be separated within half an hour at ordinary temperatures, and much more rapidly upon the application of heat."

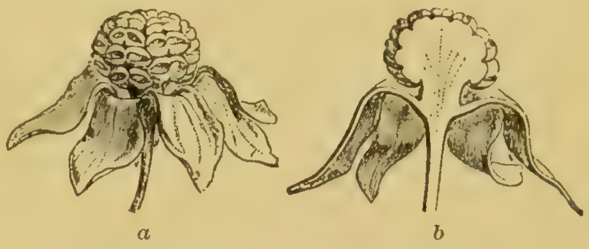

FIG. 23.—Staminate Flower of the Cocculus Indicus—*a*, entire flower; *b*, the same in section.

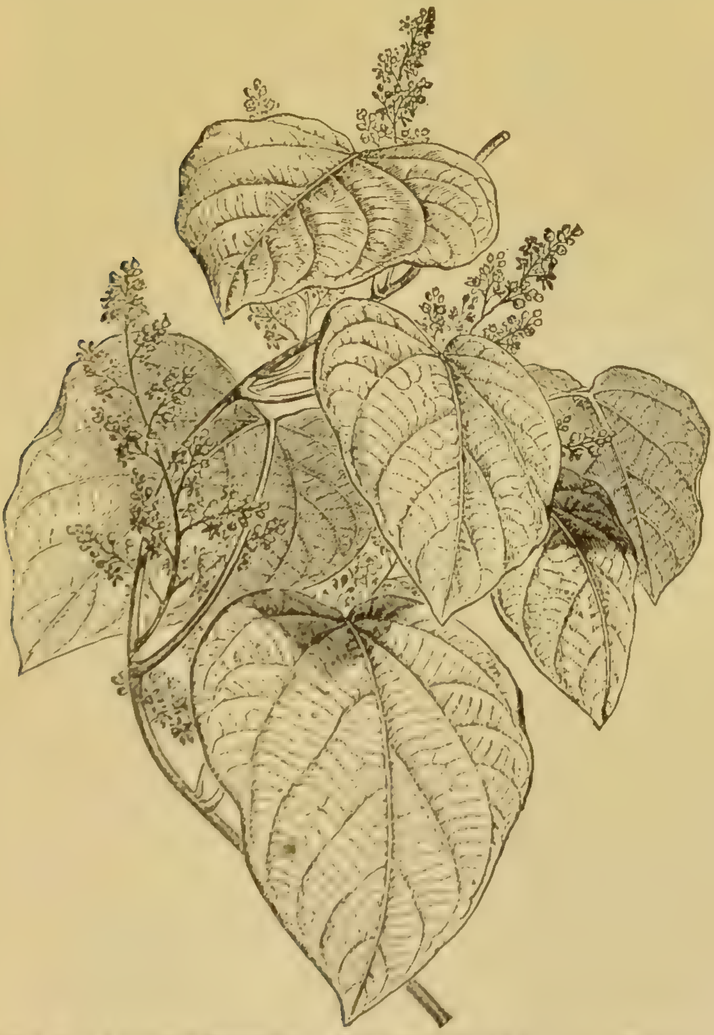

FIG. 24.—Anamirta Paniculata. Flowering (male) branch. (Baillon.)

There are no preparations of cocculus indicus, the active principle only being official. The therapeutic action of picrotoxin is that of a spinal stimulant. In poisonous doses it resembles strychnine, the motor centres being over-stimulated and ultimately paralyzed. The centres of the medulla are particularly affected, more especially the respiratory centres. Its action upon the brain is not marked, although it is commonly supposed to be an intoxicant. Its local action is severely irritant, stimulating all the secretions.

Symptoms.—In cases of poisoning there is burning in the throat and stomach, with pain, cramps, vomiting, and diarrhœa which may end in enteritis. There is incoördination of the muscles, twitchings and increased reflexes, which may end in spasms and convulsions. The respiration becomes rapid and long drawn, distending the thorax in a very characteristic manner; there may also be spasms of the glottis. The heart's action is increased in rapidity and force, at first; but as the effects of the poison advance, the presiding centre becomes exhausted, and death takes place from failure of the heart. A condition of asphyxia may be threatened and coma may precede death.

The poison is absorbed rapidly from the stomach and from the surface of the body. Its course after absorption is uncertain. The tissues of animals poisoned by picrotoxin are supposed to be poisonous to other animals. The excretion of the poison by the urine is uncertain.

Instances of poisoning are rare. In one case the application of a tincture to the scalp of a child for the purpose of destroying pediculi caused death, which was preceded by muscular twitchings, contracted pupils, and convulsions (*Medical Examiner*, Vol. VIII., 227). Two other cases are recorded. In one case several men drank of some beer that was poisonous, one of whom died. The other case was that of a boy, aged twelve years, who ate of a mixture of the berries intended for use in destroying fish; gastro-enteritis followed and terminated in death on the nineteenth day (*Medical News*, November 3d, 1866; *Philadelphia Medical Times*, Vol. XIV., 748).

The *post-mortem appearances* are of little value excepting the evidences of gastro-intestinal irritation or possibly enteritis and peritonitis.

The *treatment* consists in removing the poison from the stomach and administering chloral hydrate, which lessens the motor-excitability and spasms. Chloroform also may be required when the convulsions are severe. In experimental work paraldehyde and amyl hydrate have been found to lessen the toxic effects (*Centralblatt für klinische Medicin*, August, 1889), and should prove useful as antidotes.

Colchicum.

Colchicum autumnale, Linn., a member of the order Liliaceæ, does not occur in America, but is found growing wild in Great Britain and is indigenous to central, eastern, and southern Europe. It is closely allied to veratrum. The parts that are employed medicinally are the corm and seeds, but all parts of the plant contain the active principle. The plant is biennial, producing about September a beautiful purple flower resembling the crocus, and the following spring the leaves appear and continue to grow until midsummer when the seeds are ripened. The young corm springs from the side of the old one in midsummer and increases in size until the flower appears without foliage. The seed capsules remain underground until the succeeding year when they appear with the leaves. The foliage begins to fade in midsummer at the time that the new plant begins to grow. The parent corm begins to shrivel as the new one grows. The medicinal properties of the plant are most marked in the second season when the seeds are ripened and before the young offshoot has acquired any strength.

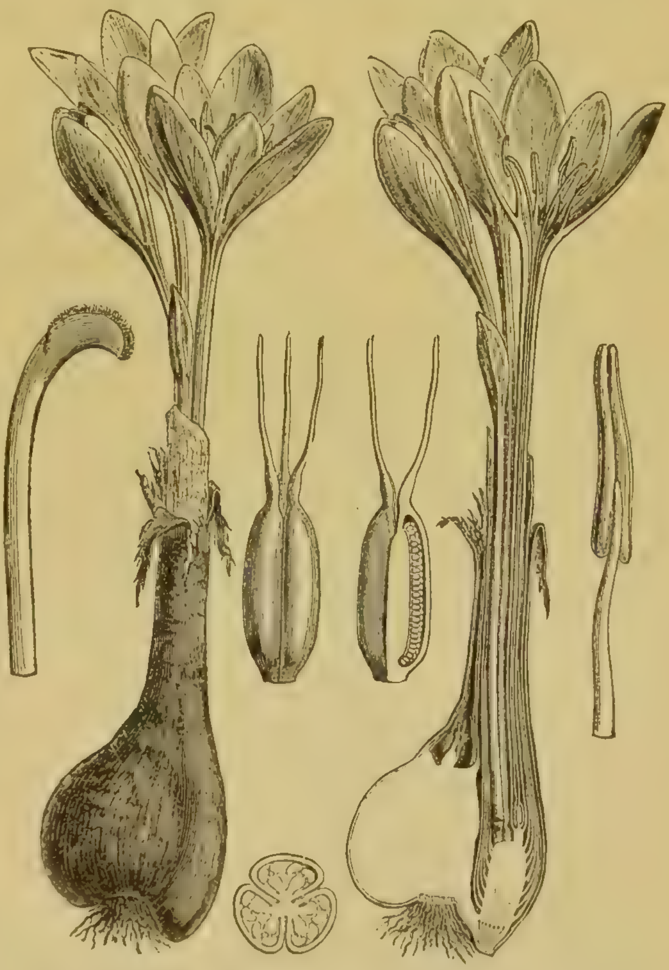

Fig. 25.—Flowering Plant of Colchicum Autumnale; one-half natural size. Showing details of structure of corms, flower, and ovary.

The corm and seeds are the parts employed in medicine. They are not now administered in substance, but have been so employed, the dose of the powdered corm or seed being from two to ten grains.

The seeds are more uniform in strength and their preparations are more reliable than those of the corm.

The United States Pharmacopœial preparations consist of a wine of the root and a wine, a fluid extract, and a tincture of the seeds. The British Pharmacopœia orders two fresh extracts, extractum colchici, 25 parts of fresh corm in 1, and extractum colchici aceticum, 18 parts in 1, and vinum colchici, 1 of dried corm in 5. The tincture is the only preparation of the seed, 1 part in 8 proof spirit.

The corms are composed chiefly of starch and moisture. By drying they lose about two-thirds of their weight.

The active principle *colchicine* is present in all parts of the plant, in the root in the proportion of from .05 to .2 per cent., and in the seed more regularly about .2 per cent. Haines in Hamilton's "Legal Medicine" gives the amount present in all parts of the plant from 1 to 1.5 per cent. Blyth quotes it in the seeds 1.15, and in the bulb 1.4 to 1.5 per cent. Colchicine is considered an alkaloid. It occurs as a yellowish-white or white amorphous powder, occasionally in crystalline needles. It is intensely bitter, soluble in all proportions of water, and in spirits, benzene, ether, chloroform, and acids. It is alkaline in reaction. Heated with acids it is decomposed, forming *colchiceine*, which crystallizes in needles, is but slightly soluble in cold water, and is much less bitter than colchicine. It possesses acid properties and combines with alkalies to form salts. It is not employed medicinally, and is of but little value except in supplying a test for colchicine.

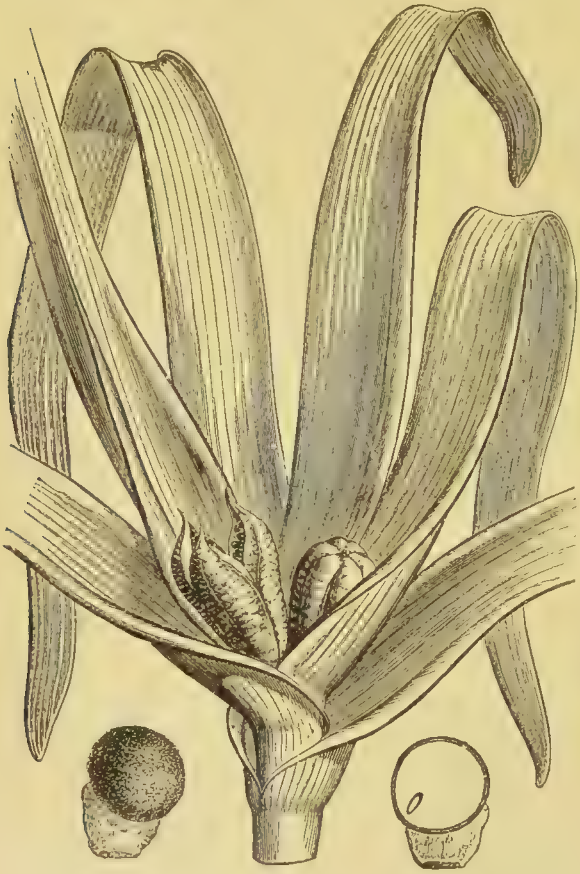

FIG. 26.—Colchicum Autumnale; showing plant in fruit. Below is seen an enlarged seed and the same in section.

The action of colchicine greatly resembles that of veratrum, and cases of poisoning, without a history of the cause, may readily be mistaken for poisoning by veratrum, hellebore, aconite, or even arsenic, or other gastro-intestinal irritant. It exerts a very severe irritant effect upon the mucous membrane of the stomach and bowels, which is supposed to be not altogether due to its local action, as the same symptoms arise when the alkaloid is administered subcutaneously. Upon the medulla and cord it is also depressant, weakening the heart and respiration and reducing reflex irritability. Sensation is also diminished. It has little or no action upon the brain.

Symptoms.—The absorption of the poison is slow and the course of the symptoms is prolonged, extending over several days and persisting after the drug is stopped. Relapses also occur frequently. The early symptoms are a burning in the throat and stomach, with nausea, vomiting, abdominal pain and tenderness, and diarrhœa. The dejecta are loaded with bile, and may contain blood, mucus, and flakes of lymph. The vomiting and purging may become excessive and be followed by collapse and death. The pulse and respiration become very much enfeebled and circulation fails, but the muscular weakness is not so early nor so severe as in poisoning by the other irritant poisons. Death does not take place for forty-eight hours or later; in numerous cases it has not occurred for a week, and in one case not until the twenty-seventh day. Convalescence is slow, the weakness and gastric disturbance continuing for some time.

Colchicum poisoning is not very common in this country, but in Europe it is of frequent occurrence from the employment both of the plant and from the drug. Of 55 cases collected (Blyth) 6 proved fatal. The fatal dose varies, as the reliability of the preparations is uncertain. Haines mentions $\frac{1}{3}$ grain of colchicine as the minimum fatal dose. Three-fifths of a grain has caused death. Taylor mentions a case in which the ingestion of three and a half drachms of the wine, in divided doses, resulted in death on the fourth day.

Five convicts, recovering from surgical operations, were given two ounces of wine of colchicum in mistake for Peruvian bark and wine. Two died at the end of eighteen hours, one in twenty, one in twenty-two, and one in twenty-nine hours. No symptoms appeared during the first two hours, except heat at the epigastrium. Then they became rapidly ill, with pain in stomach and throat, nausea, vomiting, frequent alvine evacuations, paleness, and coldness of surface, weak pulse. In six hours all the symptoms were more severe, particularly the pain in pharynx and the vomiting. The mental faculties remained unimpaired and there was no alteration in motor or sensory power. The treatment consisted in the administration of sulphuric ether and laudanum, emollient drinks, and enemata, and the application of heat to the surface and extremities. Preceding death there was lividity of the fingers and nails, an icy-cold skin, and vesical and rectal tenesmus.

Post-mortem: Mucous membranes were congested. There were no ulcers or traces of inflammation. The stomach and intestines were distended with gas, and contained a quantity of opaque fluid. No other change was present excepting a marked vascularity of the brain and cord (*L' Union Médicale*, March 27th, 1855).

The *post-mortem appearances* are limited to the gastro-intestinal tract, but in some cases where the symptoms of irritation have been severe, the local signs have not been very marked. There is generally redness of the mucous membrane and areas of ecchymosis. In advanced stages patches of inflammation and ulceration are found. These conditions are most marked in the stomach and upper parts of the intestines, but may extend into the colon. The kidneys also are sometimes congested.

The *treatment* must be chiefly directed to allaying the irritation of the stomach and bowels. The vomiting will generally have removed the poison, but if the quantity of poison has been great, the stomach-pump should be used. Tannic acid should be given to neutralize the alkaloid. Castor oil should also be given, to remove any traces of the poison from the intestines. Opiates and demulcent drinks are required to soothe the irritation. The depression will call for stimulants, and strychnine, atropine, or digitalis may prove of value to support the failing heart and circulation.

Curare.

Curare is an uncertain compound prepared by the natives of Brazil, varying according to the part of the country from which it has come and the tribes by whom it has been prepared. It is composed of the juices of several plants, but in all the secretion of some species of strychnos is to be found, that obtained from *Strychnos toxifera* being most commonly present. Curare reaches this country as a dry, brittle extract, dark brown in color, and having a disagreeable odor. In appearance and strength it varies considerably and no definite percentage of the active principle has been determined. The active principle is *curarine*, which has been separated as a crystalline body, forming in four-sided hygroscopic crystals, bitter in taste and alkaline in reaction. Pure concentrated sulphuric acid produces a beautiful blue color which distinguishes it from strychnine. Nitric acid renders it purple. Potassium bichromate and sulphuric acid cause a persistent violet color. The dose of curare is from $\frac{1}{20}$ to $\frac{1}{6}$ grain, and of curarine $\frac{1}{200}$ to $\frac{1}{100}$ grain.

Curarine acts as a depressant upon the peripheral terminations of the motor nerves, affecting first the voluntary muscles and later the involuntary. In time its action extends to the spinal cord and ultimately to the brain. Death takes place from respiratory failure.

The *symptoms* of poisoning are a feeling of lassitude and an inability to move the extremities, which extends until complete paralysis takes place. The loss of muscular power begins in the lower extremities. The respiration and heart gradually become weakened, the blood pressure falls, and a condition of cyanosis supervenes. The body temperature is increased at first, but becomes subnormal when the symptoms become severe. A glycosuria may be established. Muscular twitchings are also occasionally produced. The brain and intelligence remained unaffected until the effects of the poison are far advanced.

The course of the poisoning is rapid when the poison has been introduced subcutaneously. The symptoms begin almost immediately and within a few hours the effects begin to subside. The excretion of the poison by the kidneys is rapid and within twenty-four hours is completed. When taken into the stomach the absorption is very slow, and the poison is excreted from the system as rapidly as it passes into the blood, in consequence of which the action of the drug is never severe nor dangerous. This apparent inertness has led to the belief that the poison was decomposed in the stomach and might be taken with impunity. It has, however, been shown by experiment that if the renal arteries are ligated, the symptoms of poisoning rapidly begin and continue to increase in severity. Should any disease interfere with the function of the kidney the same result would follow.

Very severe effects have followed the accidental introduction of a few drops of a solution into a wound, but no fatal cases of poisoning are recorded.

Treatment.—If the poison has been taken into the stomach, tannic acid is to be administered and the stomach emptied by emetics and thoroughly washed. If it has entered through a wound, or has been administered hypodermically, the limb should be ligatured, the part washed, and a solution of permanganate of potassium applied with the expectation of neutralizing such of the poison as may remain unabsorbed. No antidote is likely to prove of any service in counteracting the effects of the poison, but artificial respiration should be continued to sustain respiration until the poison had been excreted. Experiments upon animals have shown that life may be retained in this manner after more than double the lethal dose has been administered. The administration of diuretics and copious draughts of water and frequent use of the catheter may hasten the excretion of the poison. Strychnine and atropine may be used to stimulate the respiration.

Digitalis.

Digitalis is a genus of the order Scrophulariaceæ, containing several species, all of which possess similar poisonous properties. *Digitalis purpurea* L., the species official in all the pharmacopœias, is indigenous to Great Britain and central and southern Europe; it is cultivated in this country for medicinal purposes and as an ornamental plant. It is a biennial, growing along hedges and woods. During the first year it consists of a bunch of rootlets and a cluster of mullien-like leaves, dark green and pubescent. In the second year it sends forth a simple leafy stalk, terminating in a long raceme of drooping, somewhat bell-shaped, purplish-pink flowers. The leaves are dark green and moderately hairy on the upper surface, and of a lighter color and very pubescent on the under surface. They are oblong, the lower ones as much as eight to twelve inches long, tapering at the base into winged foot stalks, crenate with blunt-pointed apices. The mid-rib is broad and prominent. All parts of the plant have the characteristic medicinal properties; the seeds are said to be about ten times stronger than the leaves, but the latter only are employed in medicine. They are directed to be gathered when the flowers are partially expanded, as they contain at that time the largest amount of the active principles. The cultivated is not so active as the wild plant. The dried leaves

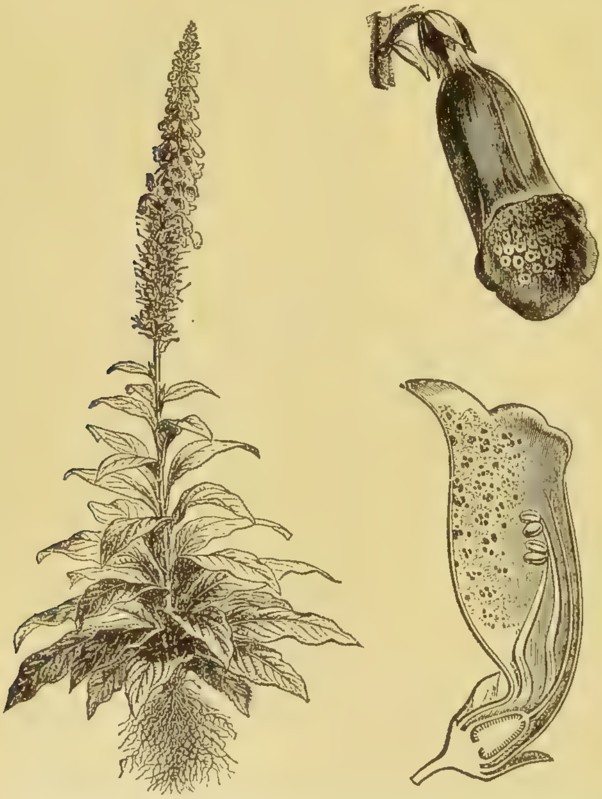

FIG. 27.—Digitalis Purpurea, Linn. Foxglove. Plant in blossom, flower slightly reduced in size, and section of flower natural size. (Baillon.)

and the powdered leaves of commerce are very unreliable, for the active principles are exceedingly unstable, they are readily injured in the process of drying the leaves, and the deterioration is increased by age and exposure. On this account digitalis and its preparations are often almost devoid of any medicinal or toxic action.

The active principles of digitalis are a series of glucosides, the chemical composition of which is very complex. The analysis of Schmiedeberg is now very generally accepted as representing these constituents. He describes four principles: (1) *Digitalin*, identical with Homolle's French digitaline, digitaline amorphe of the Codex, and digitalin of the British Pharmacopœia, formerly official in the United States Pharmacopœia. This is insoluble in water, but readily soluble in alcohol. (2) *Digitoxin*, which is essentially digitaline cristallisée of the Codex, and Nativelli's digitaline. It is insoluble in water and sparingly soluble in alcohol. (3) *Digitalein*, forming chiefly the German soluble digitalin, soluble in both alcohol and water. (4) *Digitonin*, which is devoid of the physiological action of digitalis, soluble in water and alcohol, and resembles saponin in its chemical characters. Gum, resins, and various inert substances are also present. Digitalin when obtained pure is a very active medicine, but as generally supplied is uncertain in its action and sometimes almost inert. So irregular is the strength of the different digitalins that many accidents have arisen from the substitution of one form for another in repeating prescriptions. Digitoxin is now prepared in a pure state and is coming into very general use. It is similar in its properties to digitalin but much more active, the dose being from $\frac{7}{200}$ to $\frac{7}{100}$ grain.

Digitalis, in small doses, is a cardiac stimulant, and in toxic doses a paralyzant of that organ, which effects are produced through its action upon the nerve centres presiding over the heart and circulation. In its primary action, a stimulation of the inhibitory nerves produces a slowing of the heart, and a similar action upon the intrinsic ganglia prolongs and increases the force of the contraction. At the same time the vaso-motors are stimulated and the blood pressure is raised. The secondary action, arising from overdoses or the prolonged use of the drug, is to exhaust these centres and cause a weakening of the heart, a relaxation of the vaso-motors, and death from syncope or asphyxia. The ill effects of the drug also extend to the other centres of the medulla, to the spinal cord, and to the brain.

The action of digitalis upon the gastro-intestinal tract is as characteristic as that upon the heart. It irritates the mucous membrane and stimulates the secretion of the glands of the mouth and stomach, and later irritates the small and large intestines.

The *symptoms* of poisoning begin as a sense of heat and pain at the epigastrium with nausea and vomiting, followed sometimes by salivation, irritation of the throat, colic, diarrhœa, and other disturbances of the stomach and bowels. These symptoms are not all due to its local irritant action, but in part arise from central disturbance; this is particularly true of the nausea and vomiting. There also occur muscular weakness, a lowering of temperature with chilliness and shivering, disturbances of vision, noises in the ears, and a sense of weakness, depression, and uneasiness. The heart is at first slow and tumultuous, but gradually becomes spasmodic in its action, and ultimately increases in rapidity, with irregularity and feebleness. The respiratory disturbance which accompanies this depression and the consequent deterioration of the blood add a condition of cyanosis to the direct poisonous effects. During the last stages the accumulation of carbonic acid becomes very marked and may give rise to convulsions and coma. The action of the kidneys, which at first was increased, becomes suppressed. The intellect generally remains clear until the last, or until influenced by the impure blood. The action of digitalis is greatly increased by certain diseased states of the heart, which has enfeebled the muscular tissue, such as dilatation and fatty degeneration. When there is disease of the arteries, or other organs to interfere with circulation, or when ascites or general anasarca is present, the unfavorable effects are more rapidly developed.

Digitalis is absorbed from the stomach, intestines, rectum, and denuded surfaces of the body; its passage into the blood is slow, and from one-half to two or three hours may elapse before the symptoms appear. Its course after absorption is undetermined, as the glucosides are rapidly decomposed and cannot be detected in the tissues of the body. Its excretion is also uncertain and its presence in the urine has not been clearly demonstrated. The continuous use of moderate doses tends to an accumulation of the drug in the system, and in time causes depression, nausea and vomiting, disordered heart, and other evidence of its toxic action. The question of cumulative action of the drug, manifested by a sudden onset of toxic symptoms, is one that has received much attention and is doubted by many. During the administration of digitalis the occurrence of acute disease, a sudden checking of the action of the kidneys, or the rapid removal of ascitic fluid may immediately be followed by serious circulatory depression and tumultuous action of the heart; but any sudden action without such causes is not so evident. Numerous instances have been reported of unfavorable symptoms arising during its prolonged use and persisting after the drug has been stopped, but in all such cases there have been signs of a gradually increasing action until the limits of safety have been passed. It will generally be found that the symptoms of poisoning have been preceded by premonitory signs.

The fatal quantity is uncertain on account of the variability of the drug and the incompleteness of the reports of fatal cases. A case is reported of a woman, 22 years of age, who took by mistake an infusion prepared from 2 drachms of the leaves, and died on the fifth day (Ziemssen). Blyth gives the following table of maximum doses and considers that double the quantity would be dangerous—powdered leaves $4\frac{1}{2}$ grains, infusion 480 minims, tincture 45 minims, digitalin $\frac{1}{33}$ grain. Haines (Hamilton's "System of Legal Medicine") gives 8 or 10 grains of the powdered leaf as the probable fatal quantity. These doses are far below the line of danger according to present uses of the drug. Pareira refers to drachm doses of the tincture three times a day for a prolonged period, and to doses of one ounce of the tincture to be repeated in twenty-four hours. It has been very common to treat delirium tremens with half-ounce doses of the tincture, and more recently pneumonia has been treated by doses of the infusion or juice which represents 125 grains of the leaves. Of digitalin also very large doses have been given.

A man, 22 years of age, to escape military service, took during four weeks about 211 grains of powdered digitalis leaves. After eleven days he became ill with nausea, loss of appetite, tenderness over the stomach, pain in the head, giddiness; the pulse was 56, temperature normal. Before death the pulse became 52, with vomiting, noises in the ears, disordered vision, great debility; he had frequent fainting fits, and died suddenly after an attempt to walk from his bedroom (Blyth, *Vierteljahrschrift für gerichtliche Medicin*, 1876, 402).

A woman, 23 years old, took, with suicidal intent, 16 granules of digitalin at 7 A.M.; two hours later shivering and giddiness and hallucinations began and continued throughout the day and night, with dyspnœa and cold sweats. The following morning all symptoms subsided. Forty granules were then taken and in one hour she became giddy, and suffered with chilliness, cold sweats, copious vomiting, colicky pains, and muscular weakness. No action of the bowels; urine was suppressed; the eyes felt large and bulging. This condition continued for nearly forty-eight hours when she was seen by a physician; she was then motionless, and had exophthalmus, dilated pupils, pallor, cold sweat, pulse small and intermittent, almost imperceptible, 46 to 48 per minute; epigastrium tender. On the third day the symptoms began to improve and recovery followed (Blyth).

Du Koppe for experiment took of digitoxin 1.5 milligrammes and the next day 1 milligramme without result. Four days later he took 2 milligrammes in alcoholic solution. An hour later he felt faint and ill and giddy, pulse normal in frequency but irregular. Four hours later the pulse was 58 per minute and intermittent, vomiting commenced, and the pulse sank to 40, there was a feeling of constriction and uneasiness in the chest with every intermission. Muscular power reduced, disordered vision, sleeplessness. On the third day he began to improve but did not recover for several days (Blyth, *Archiv für Experimentelle Pathologie und Pharmakologie*, Vol. III., 239, 1875).

The *prognosis* of digitalis poisoning is generally hopeful, as the tardy absorption allows treatment to be more effectual than in many other forms of poisoning.

The *treatment* consists in emptying and cleansing the stomach, administering tannic acid to prevent absorption, and purgatives to remove any traces of poison from the stomach and bowels. To counteract the depressant action stimulants are indicated. Warmth to the extremities and surface, electricity, and other such means are required to maintain the circulation. Rest in the recumbent position should be maintained for the same purpose and to prevent any sudden overtaxing of the heart. The gastric pain and irritation will be relieved by opium. No physiological antidote has proved of any service in this form of poisoning.

Aconite has been suggested as an antagonist to the action of digitalis upon the circulation, as in small doses the action of the two is opposite in character, and digitalis has been used in cases of poisoning by aconite. Aconite, however, would require to be used with caution on account of its depressant effects and in certain stages would aggravate the condition of the patient. When there is danger of failure of the heart and circulation, strychnine and other cardiac stimulants will prove of service.

The *post-mortem examination* furnishes no evidence as to the cause of death unless traces of the poison are discovered in the stomach and intestines. In some cases of poisoning by the leaves, areas of congestion have been found on the mucous membrane of the stomach. The vessels of the brain have also been described as greatly congested, but in other instances the brain has been anæmic. The other organs are generally in a condition such as might be expected in death from asphyxia.

As the glucosides are decomposed in the system no evidence of any value is to be derived from an examination of the organs, although, in the famous Pommerais trial, evidence of the presence of some heart poison was said to have been obtained by physiological tests after the body had been buried thirteen days. In this case the same effects were derived from tests with vomited matter obtained from the clothes and in the bedroom of the patient. The presence of unabsorbed portions of the poison will furnish the most trustworthy evidence, and all vomited matter and the contents of the stomach should be preserved for the purpose. A further difficulty in determining the presence of this poison is the absence of any trustworthy chemical tests.

Gelsemium.

Gelsemium sempervirens, L., *Gelsemium nitidum*, Michx., the yellow jasmine, order Loganaceæ, is a beautiful flowering plant indigenous to the United States from Pennsylvania southward, and introduced into the northern States and Europe as an ornamental plant. It is an evergreen climbing plant with a slender, smooth, purplish stem and dark shining green leaves, lanceolate and entire, with minute stipules, growing opposite to each other and bearing in their axils clusters of flowers, bright yellow in color and sweet scented. The corolla is tubular with a fine-lobed border, one to one and a half inches long. The fruit is a drooping pod. The stem springs from a large woody rhizome, supplied with numerous fine rootlets. The rhizome and roots are official under the title gelsemium. They are cylindrical, ½ to 1 inch long, ¼ to ¾ inch thick, yellowish-brown externally, with dark purple longitudinal lines. The bark is thin and closely attached. The properties of the plant depend upon the alkaloid *gelsemine*, which is present in all parts of the plant, but is most abundant in the bark of the rhizome, estimated at about ¼ to ½ of 1 per cent. (Wormley). It is a yellowish-white amorphous powder, extremely bitter, very soluble in chloroform and ether, less so in alcohol, and only slightly soluble in water. Dose $\frac{1}{60}$ to $\frac{1}{30}$ grain. Gelsemine exists in the plant in combination with gelsemic acid.

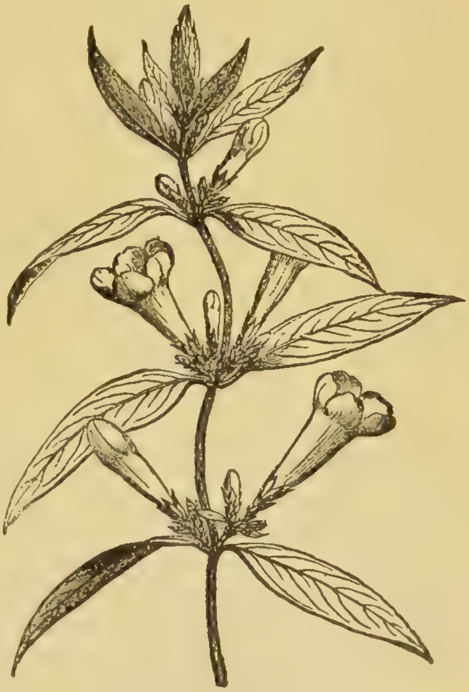

FIG. 28.—Gelsemium Sempervirens, Yellow Jasmine or Jessamine, of the Southern United States.

Gelsemine is a motor paralyzant, its action being directed to the motor centres of the cord, in this way differing from curarine and coniine, in which the motor paralysis is due to the peripheral terminations of the nerves being affected. The respiratory centre is specially depressed, the respirations growing weaker and weaker until paralysis and death take place. The heart is also depressed and the blood pressure lowered, but not so severely. Sensation is slowly influenced and the brain also becomes affected. The effect upon the eye and muscles of the face is very characteristic, the third nerve becoming paralyzed early. The muscles of the tongue, pharynx, and lower jaw are affected early.

The *symptoms* of poisoning are depression and lassitude, the limbs become weak and unable to sustain the body; tetanic spasms sometimes are present, and alternate with the muscular paresis. As the effects increase there are vertigo and dizziness, the vision becomes disturbed, the pupils are dilated, ptosis may be present, the tongue feels thick, speech becomes difficult, and the lower jaw falls and remains open. The respiration is slowed from the first and each respiratory act is an effort. Cyanosis usually precedes death. When the symptoms are severe sensation is affected and complete anæsthesia may be present.

The course is rapid, in some cases death taking place in one or two hours. The symptoms begin early, and the effects of the drug begin to pass off in a few hours if the tendency is to recovery.

The smallest quantity that may cause death is uncertain; professor Wormley estimates about one-tenth grain of the alkaloid. One drachm of the fluid extract has caused death in an adult (Haines), twelve minims proved fatal to a boy three years old; thirty-five drops of the tincture caused death in one and one-half hours, and fifteen grains of a resinoid extract proved fatal to a woman in one hour (United States Dispensatory, 1890).

The *treatment* consists in removing all traces of the poison from the stomach, and employing artificial respiration. Atropine, strychnine, and nitroglycerin have been suggested as antidotes. Rest is necessary and heat must be supplied to the surface and extremities.

A man, aged 28 years, when intoxicated drank two ounces of the tincture of gelsemium. When seen his face was flushed; the pupils were slightly dilated, but reacted to light, and there was slight ptosis. At the end of an hour he took half an ounce of the fluid extract, and when found twenty-five minutes later was conscious and able to speak; the limbs were relaxed and the face was pale. Free vomiting was obtained, but the man rapidly became unconscious and fell into a collapse; the pulse was 130, respirations 40, entirely thoracic; the pupils were moderately dilated and reacted to light. Injections of brandy and the application of the faradic current caused improvement for a few moments, but the man relapsed quickly and died in five hours. Atropine, carbonate of ammonia, and amyl nitrite were employed (*Boston Medical and Surgical Journal*, Dec. 22d, 1881).

There are no characteristic *post-mortem signs*, but the alkaloid and the gelsemic acid may be detected in the tissues, and furnish decided evidence of the cause of death. Both resist decomposition and have been detected four and a half months after death.

Hellebore.

Helleborus is a genus of Ranunculaceæ, closely allied to aconite. It is indigenous to central and southern Europe, and certain species occur in Great Britain. The best known are *H. niger*, the Christmas rose, *H. fœtidus*, and *H. viridis*. Black hellebore is recognized in the German Pharmacopœia and was formerly official in the United States Pharmacopœia and in the British Pharmacopœia. Its use as a popular medicine is still in vogue in the country districts of England and many instances of poisoning have occurred. H. fœtidus is extensively employed as a remedy for worms.

Black hellebore is a perennial herb with a thick, branched, horizontal rhizome, with many long rootlets; the leaves spring in a cluster from the stalk, four to eight inches long, palmately divided. The flowers are large, on bracted terminal scapes; sepals are rounded or ovate-obtuse, large, white, slightly tinged with pink, eventually becoming green; petals are small, tubular, and green. Fruit forms into compound pods, with numerous black and shining seeds.

The active principles have been shown to be two glucosides, *helleborin* and *helleborein*, both of which have similar action. Hellebore is strongly irritant to the mucous membranes and has an action upon the heart resembling digitalis. The pulse is primarily reduced in frequency and its force is increased. In overdoses, however, it produces a rapid and feeble action and causes a sudden failure, respiration continuing after the heart has ceased to beat. The secretions generally are increased. It also acts upon the nervous system, producing at first a stimulating effect upon the cord which is followed by debility, paralysis of motion and sensation, with cerebral disturbances.

The *symptoms* of poisoning are very similar to those of veratrum and the two poisons may readily be mistaken one for the other. The local irritant action upon the mucous membranes causes heat and burning of the mouth and stomach, nausea, vomiting, salivation, and diarrhœa. The primary stimulating action upon the motor centres and heart may not appear and the general depression may then be very marked, with muscular twitchings, paralysis, dilated pupils, and in time failure of heart and circulation. The cerebral disturbance is more marked than in poisoning with veratrum, producing blindness, loss of hearing, anæsthesia, convulsions, and coma.

The quantity that is liable to cause death is very uncertain. Pareira mentions ten to twenty grains of the powdered root as a drastic purgative. In one case a teacupful of an infusion, made from one and a half ounces of the root to ten ounces of water, caused dysphagia, pain in the stomach, salivation, and general irritation of the intestinal tract, with severe prostration approaching to collapse. The treatment consisted of emetics of sulphate of zinc, copious draughts of hot water, camphor, coffee, and the yolk of egg. Heat was applied to the surface. The patient rallied in three hours, and improved steadily from that time (*Lancet*, 1856, II., 100).

The *treatment* consists in removing from the stomach all traces of the poison and administering the same remedies as in poisoning by veratrum.

Hemlock.

Conium maculatum, L., poison hemlock, order Umbelliferæ, is found very generally throughout the United States and Canada, where it has been introduced from Europe. It grows in open waste places and is very common in the neighborhood of towns and villages. All parts of the plant are poisonous and numerous accidents have arisen from its mistaken use. It flowers in June and July, exhaling a disagreeable odor, which, if inhaled for some time, is said to produce narcotic effects. The plant is most active in hot, dry seasons. The root is biennial, long, fleshy, simple, or forked, yellowish-white; the stem is simple, erect, two to six feet high, smooth, somewhat glucous, shining, with numerous dark purple spots; leaves are tripinnate, triangular in outline, dark, shining green, with long furrowed petioles, sheathed at their base. The flowers are in umbels, numerous, with a general involucre of several bracts, and a partial involucre of three bracts on one side. Corolla consists of fine white sepals, obcordate, with inflexed points. Fruit, one-eighth inch long, is broadly ovate, compressed laterally, with undulated crenated ridges, the inner surface of the seed has a narrow longitudinal groove. The distinctive points are the spotted stem, shining leaves, involucres, and fruit. The whole plant emits a disagreeable, nauseous odor when bruised.

The fruit only is official in the United States; in Great Britain both the leaves and fruit are recognized. The quantity of the alkaloid ranges from one-half to two per cent. The fruit is considered the most active part of the plant, the leaves less so, and the root and stem very uncertain. All parts of the plant are, however, unreliable in their strength, and the preparations are very variable.

The active principle is *conine*, a yellowish, oily alkaloid. It is volatile, readily decomposed by heat and exposure. It possesses the disagreeable odor of the plant, is soluble in water (about one part in one hundred), very soluble in alcohol. Other constituents are *methylconiine, conhydrine, coniic acid,* and a volatile oil. They are present in coniine in varying quantities, but have but little influence upon the properties of the coniine as their action is similar. Methylconiine varies somewhat, attacking the motor centres early and lessening reflex action.

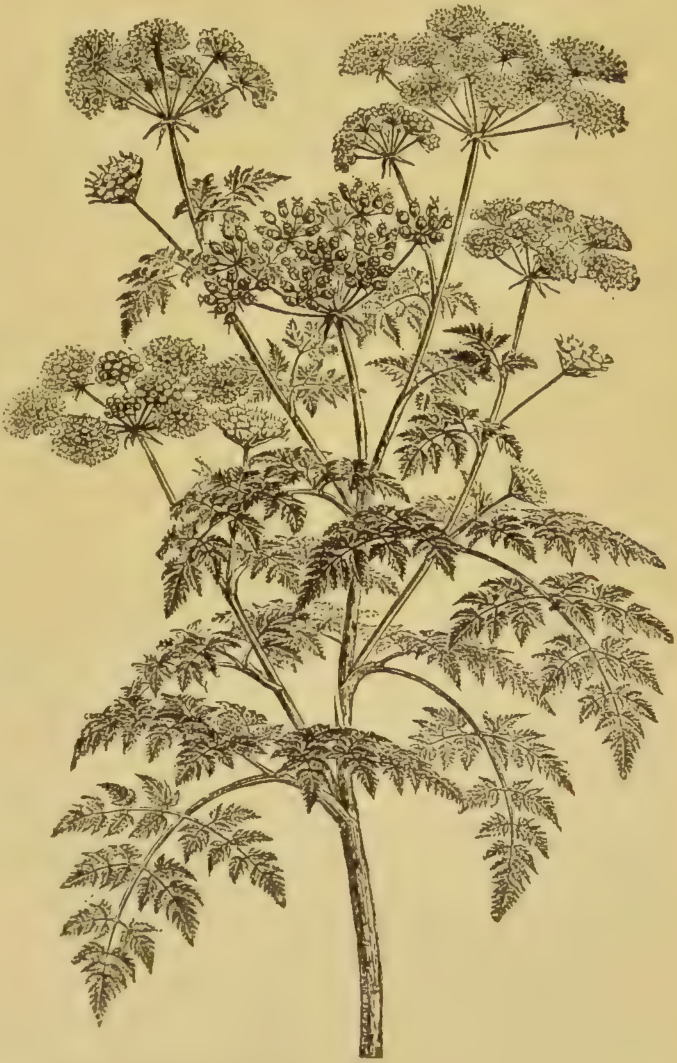

FIG. 29.—Conium Maculatum, Hemlock. Flowering plant.

Coniine resembles curarine in its action, producing motor depression by paralyzing the periphery of the motor nerves. Its effect extends to the motor centres and spinal centres generally. Sensation is dulled early, and the absence of common sensibility becomes very marked as the poisoning advances. The muscles retain their irritability, and the convolutions are uninfluenced. The respiratory organs are especially depressed and in fatal cases death is due to respiratory paralysis. The heart is also depressed but not in so marked a degree. It produces a local irritant action upon the stomach with heat and burning, and nausea; vomiting, however, is sometimes difficult to obtain.

The symptoms of poisoning are a feeling of general weakness and inability to move, with more or less numbness, the vision becomes disturbed, eyelids droop, there is difficulty in articulating, and a sense of constriction of the chest, with an effort to breathe, and feeble heart. This condition increases until complete paralysis is present and a condition of cyanosis arises.

As the absorption of the poison is rapid and its excretion with the urine begins early, the course of a case of poisoning is of short duration. When the quantity of poisoning is large, the early symptoms may be almost unnoticed and a condition of complete paralysis appear at once.

The dose of coniine is $\frac{1}{60}$ to $\frac{1}{10}$ grain. One grain or one drop is considered a dangerous quantity and liable to prove fatal. The drug is very uncertain in its quality and may sometimes be taken in much larger doses without serious results. Fifteen drops of the alkaloid, and 320 drops of the alkaloid, in divided doses, during six or seven hours, have proved fatal.

The post-mortem appearances furnish no evidence of the cause of death, unless traces of the poison are to be found in the stomach. The characterisic odor may be detected. The alkaloid is excreted unchanged, and may be detected in the urine. The volatile character of the alkaloid makes it doubtful if it remains unchanged in the tissues for any length of time.

A man ate a large quantity of hemlock leaves in mistake for parsley. In from fifteen to twenty minutes there was loss of power in the lower extremities, but he apparently suffered no pain. In walking he staggered as if he was drunk; at length his limbs refused to support him and he fell. On being raised, his legs dragged after him, and when his arms were lifted they fell like inert masses and remained immovable. There was perfect paralysis of the upper and lower extremities within two hours after he had taken the poison. There was a loss of the power of swallowing and a partial paralysis of sensation, but no convulsions, only slight occasional motions of the left leg; the pupils were fixed. Three hours after eating the hemlock the respiratory movements had ceased and death followed from asphxyia (Taylor, *Edinburgh Medical and Surgical Journal*, July, 1845).

The *treatment*, as in poisoning by the other motor-depressants, consists in removing all traces of the poison from the stomach, promoting its elimination, and maintaining the respiration until the effects have passed. The use of emetics and the stomach-pump, assisted by tannic acid, secures the first indication, and copious draughts of hot water, with heat to the surface, the second. When the respiration fails it may be kept up by artificial means, and the accumulation of carbonic acid in the blood prevented by inhalations of oxygen. Electricity is also recommended. As antidotes, atropine and strychnine are suggested as stimulants to the motor centres.

Closely allied to conium are several other genera, the members of which are more or less poisonous, and are frequently the cause of serious accidents. Of these the most common are the various species of cicuta or water hemlock; sium or water parsnip, æthusa or fool's parsley, and œnanthe or wild parsnip. The species of cicuta and œnanthe are the most active; the latter, however, is not found in America. There appears to be some uncertainty regarding the poisonous properties of the other plants. *Æthusa cynapium* has received some attention from Dr. Harley (St. Thomas's Hospital Reports, 1875) who declares it to be harmless. *Cicuta virosa*, L., of Europe is represented in this country by *C. maculata*, L., which resembles it so closely that it is now considered simply a variety of the European plant. Its toxic properties are equally powerful and its active constituent is the same. It is a coarse perennial, growing three to six feet high, in swamps and damp places, very common throughout the whole of America. The stem is streaked with purple, bearing thrice-pinnately compound leaves, the flowers and fruit forming in numerous large umbels. The plant is distinguished by the fruit, which is smooth, flattened laterally, and equally five-ribbed. All parts of the plant are poisonous, but the root is the most active. Some animals, as the sheep and goat, are said to eat the leaves with impunity.

The active principles are a resinous substance named *cicutoxin*, and a volatile alkaloid *cicutine*, which resembles coniine. The latter is of little importance. Cicutoxin is thought to resemble picrotoxin in its physiological action, as it is a powerful motor stimulant, acting especially upon the medulla. Following its stimulation is a period of depression and paralysis which ends in death. A great deal of experimental work has been done and the effects of cicutoxin carefully noted (Blyth). Upon animals it caused rigidity of the muscles of the limbs and abdomen, tetanic convulsions, paresis of the extremities, and finally paralysis and death. Diarrhœa and salivation also occur. The lethal dose for cats was $\frac{1}{6}$ grain for each 2.7 pounds. Analysis of the cases of poisoning in man show the chief symptoms to be pain and burning in the stomach, nausea, vomiting, and convulsions, in some cases coma. Death may take place in a few hours.

Hyoscyamus.

Hyoscyamus niger, L., order Solanaceæ, is indigenous to Europe and eastern Asia, but has been introduced into America where it has become a common roadside herb in the North and East. It is a clammy, pubescent, fetid biennial, sometimes annual plant, growing from one to three feet high. The leaves are large, coarse, triangular-ovate, clasping, sinuate, and toothed at their margin. The flowers are yellow with purplish veins, springing from the axils of the bracts in a terminal crowded spike. The fruit is a capsule from which a lid falls allowing the seeds to escape at maturity. The root is simple, large, and fleshy. The first year's growth of the plant, when biennial, consists only of radial stalked leaves, no stem being formed.

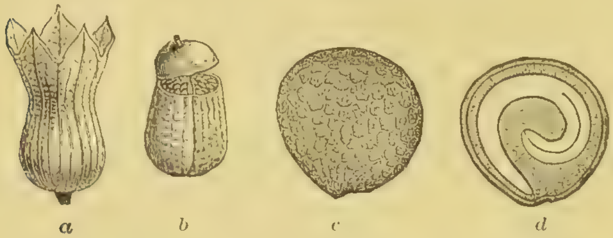

Fig. 30.—Hyoscyamus Niger, Henbane: flower, fruit and seed. *a*, Flower; *b*, fruit, with lid open showing seeds; *c*, seed, enlarged; *d*, the same in section, showing embryo coiled.

All parts of the plant are active, its properties being most marked in the second year, as the flowers expand, at which period the leaves and flowering tops are directed to be collected for official use. After inflorescence the percentage of alkaloids is much less, the seeds being the strongest.

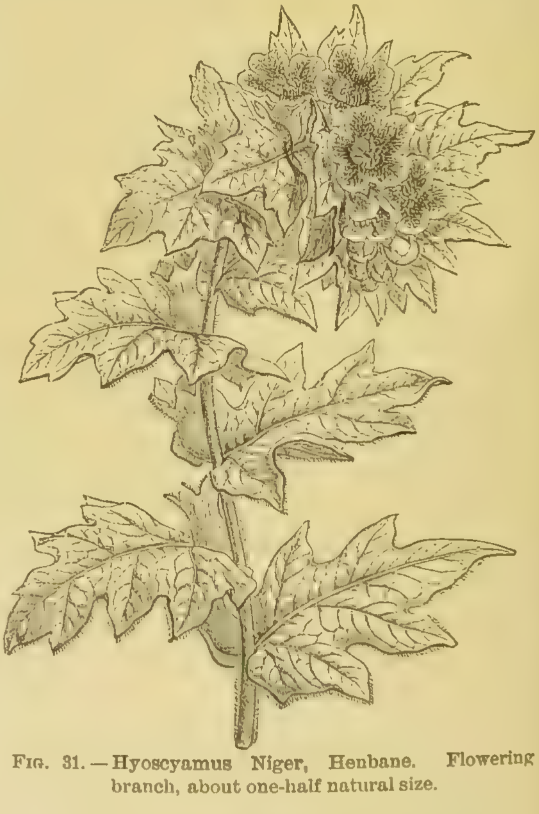

Fig. 31.—Hyoscyamus Niger, Henbane. Flowering branch, about one-half natural size.

The alkaloids are *hyoscyamine* and *hyoscine* which are isomeric, having the formula $C_{17}H_{23}NO_3$, the former being obtained in colorless, transparent needles, with an acrid, disagreeable taste, but slightly soluble in water, very soluble in acidulated water and alcohol. Hyoscine is a syrupy liquid, forming crystalline salts with acids. It was formerly obtained as an amorphous mass known as amorphous hyoscyamine. The salts of the alkaloids are generally employed, the hydrobromate of hyoscine, and the hydrobromate and sulphate of hyoscyamine being official.

The preparations of the United States Pharmacopœia and British Pharmacopœia vary somewhat in strength. The extract of the United

States Pharmacopœia is administered in doses of $\frac{1}{2}$ to 1 grain, that of the British Pharmacopœia of from 5 to 10 grains, the latter being a green extract. The fluid extract of this country is also much stronger, the dose being 5 to 20 minims, that of the British Pharmacopœia $\frac{1}{2}$ to 1 drachm. The tinctures are very similar in strength.

The *action* of hyoscyamus has always been recognized as being similar to that of belladonna, the points of difference being the more marked calmative and soporific action of the former and the greater strength of the latter drug and its preparations. This is more clearly understood now that the identity of hyoscyamine and atropine is accepted, any difference in the drugs being explained by the presence of hyoscine. The action of hyoscine is very decidedly depressant to the cerebral, respiratory, cardiac, and motor centres generally. It produces profound coma and great weakness of the respiration, which may become "Cheyne-Stokes" in character; the skin may be moist and covered with perspiration. In other respects its actions and mode of causing death are the same as that of atropine. Cases of poisoning by the alkaloids are very common, but a fatal termination is unusual, the tendency being to recovery. In some instances very small doses have given rise to alarming symptoms. This is particularly noticeable in certain neurotic patients in whom the stability of the nervous system has been impaired. In cases of poisoning by the growing plant or the preparation, the diagnosis from that of belladonna or other members of the Solanum family may be very difficult.

A man, 50 years old, was given $\frac{1}{6}$ grain hyoscine hydrobromate during a maniacal attack. In two or three minutes he fell to the ground; he was pale, the pupils were dilated, the breathing was difficult and stertorous, the limbs were contracted spasmodically; the pulse was weak, the temperature subnormal. Hypodermics of ether, 20 minims every half-hour, were given. In one and one-half hours the spasms relaxed, the symptoms began to subside, and within five or six hours the man became conscious and was able to converse (*New York Medical Journal*, April 28th, 1894).

An adult female took $1\frac{1}{2}$ ounces of tincture of hyoscyamus in mistake for black draught. In ten minutes she had a burning and tingling sensation in the hands, feet, and legs; she became giddy and delirious, with dry throat. The legs soon became powerless; a purplish rash appeared over the body; the woman gradually became insensible; the pupils were dilated. In three hours the bowels were moved, the motion smelling strongly of the drug. In twenty-four hours she was able to articulate but the symptoms remained for six days (*Lancet*, July 5th, 1873).

Adult, aged 40, had insomnia from business troubles and drink, and was given $\frac{1}{100}$ grain hyoscine hydrobromate hypodermically in the evening and repeated in forty-five minutes; very slight effect was produced. The following morning $\frac{1}{50}$ grain was given. In fifteen minutes the patient complained of feeling numb and peculiar; his hands became purplish, and in attempting to walk his legs gave way and he fell; the speech became confused and the voice was husky; pulse 90. In two and one-half hours the symptoms had passed away, but there had been no sleep or rest. In the evening $\frac{1}{50}$ grain was again given with the same result. Motor paralysis and inability to move or articulate; respiration labored and irregular; pulse 112; hands and face purplish. In three hours he had recovered. No treatment (*Medical Record,* July 29th, 1893).

The *treatment* is the same as for belladonna poisoning. In mild cases there is little to do except to remove from the stomach any poison that may continue to be absorbed. In the more severe cases the antidotal drugs will have to be employed and efforts must be made to sustain the enfeebled respiration.

Opium.

Papaver somniferum, Linn., the official source of opium, is indigenous to Asia Minor, but has spread throughout Asia and Europe. It is cultivated extensively in Great Britain and in America, and is frequently found in a wild state, having escaped from cultivation. There are numerous species of the poppy, all of which possess the same narcotic properties, many of them being very common in Europe; *P. Rhœas,* L., *P. dubium,* L., and *P. argemone,* L., being the best known. In this country they are only occasionally met with. *P. nudicaule,* L., is an Arctic species found in great abundance in Labrador, Greenland, and throughout the Arctic regions.

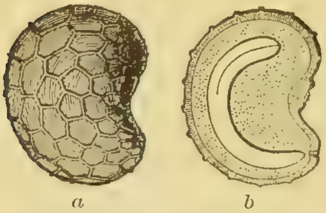

FIG. 32.—Seed of the Poppy—*a,* entire seed; *b,* section of seed showing the embryo. Enlarged.

P. somniferum is an annual herb, growing from two to four feet high, with an erect, simple stem, smooth, or with a few hairs toward the upper extremity. Leaves oblong, large, sessile, or clasping, smooth; margins wavy and incised; teeth sometimes tipped with a rigid hair. Flowers are generally solitary, from the upper axils terminal, white, in *var. nigrum,* of a purple or red color. Calyx, two green sepals, which fall as the corolla expands. Corolla, four bright, rounded petals, which also fall early. The ovary is large, rounded, and fleshy, one-celled, with a number of parietal placentæ. Seeds numerous. The whole plant, especially the capsule, is richly supplied with lactiferous vessels, which are distended with the juice at the period of ripening.

All parts of the plant contain the active principle, and cases of poisoning have occurred from the use of the leaves, seeds, and capsules of the official species, and from eating the flowers and capsules of *P. Rhœas* (Ziemssen). The young plants are said to be devoid of any narcotic property. The active principles are present in the juice of the plant, which is very abundant especially during the period of inflorescence. The opium of commerce consists almost entirely of the juice with a small proportion of foreign matter, in the form of vegetable tissue, gathered with the juice. Grosser adulterations are rarely met with in the Smyrna and better classes of opium. In the Turkey and other poor grades the impurity may be very evident.

The alkaloids comprise from one-sixth to one-third of opium, the remaining constituents being water, gum, resins, vegetable fats, and other organic compounds. The alkaloids and organic principles are exceedingly numerous and their number is constantly being increased. About thirty have been described and named, but many are undoubtedly the products of changes taking place in the mass, as they are not to be detected in the fresh juice of the poppy. With the exception of *thebaine* and *paparerine*, the physiological action of all is of a paralyzing nature. The action of these two is of an opposite character

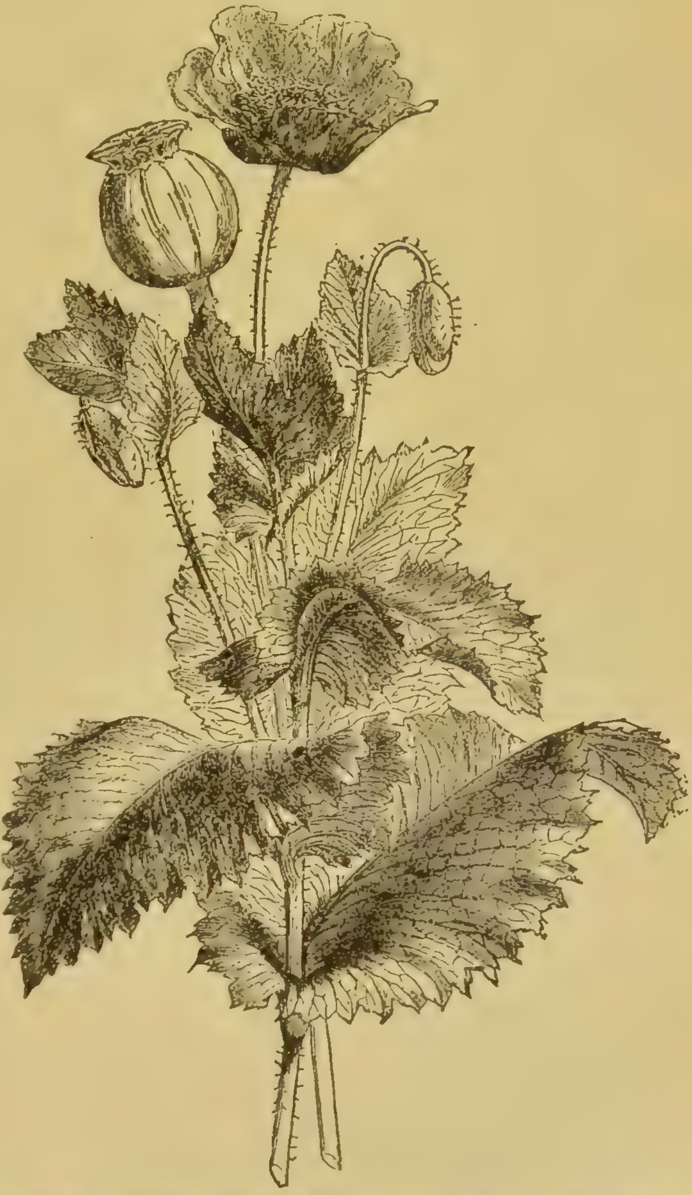

FIG. 33. Papaver Somniferum, the Opium Poppy. Flowering branch, showing flower and capsule. Much reduced.

as they possess tetanizing powers analogous to strychnine. Being present in very minute quantities they do not exert any appreciable influence upon the effects of opium. *Narcotine*, $C_{22}H_{23}NO_7$, possesses little or no medicinal action, but is of some importance on account of the large proportion present, as, together with morphine, it comprises almost the entire bulk of the alkaloids. Its prevalence, however, has been found to be in inverse ratio to that of morphine. In Smyrna and other good specimens of opium it is least abundant, while in specimens poor in morphine it is present in large amounts.

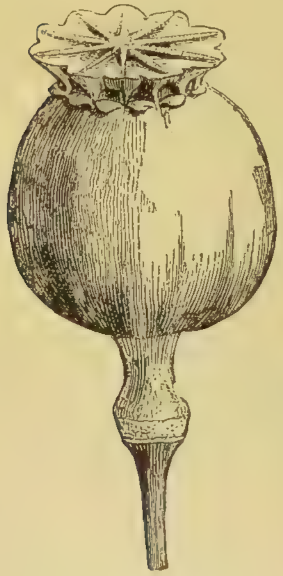

FIG. 34.—Capsule of the Poppy. About one-half natural size.

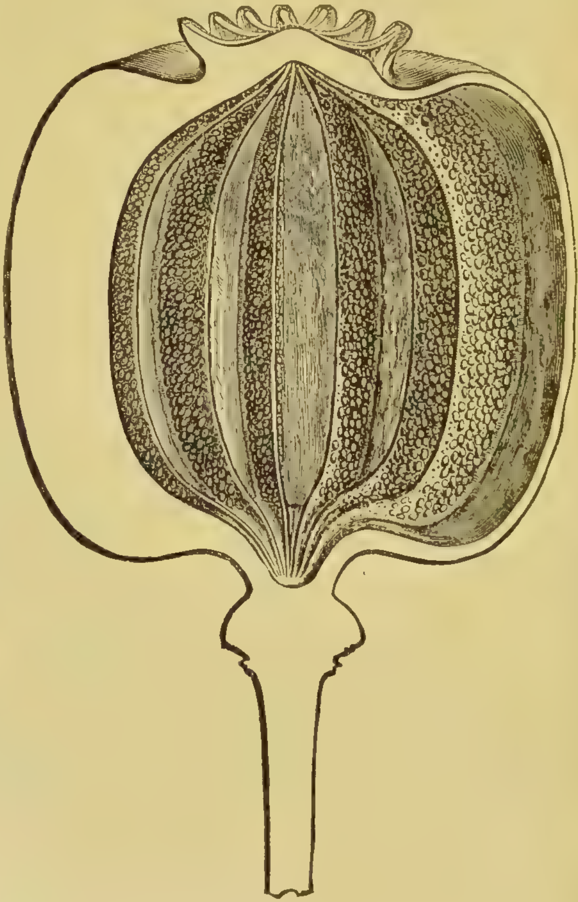

FIG. 35.—Longitudinal Section of the Capsule of the White Poppy, Showing the Wing-like Placenta.

The strength of opium is dependent upon the percentage of *morphine*, $C_{17}H_{19}NO_3$, that it contains. The amount varies greatly. The United States Pharmacopœia requires thirteen to fifteen per cent. in powdered opium, the British Pharmacopœia ten per cent. Specimens have been examined from which as much as twenty-one per cent. was obtained. The average is below rather than above the official test, and as little as five per cent. is very common in the poorer varieties of opium. The acetate, hydrochlorate, and sulphate are the official salts. They are alike in strength but differ in their solubility and physical characters.

The preparations of opium and morphine do not vary much in the various pharmacopœias, the importance of a similar strength being very evident. The various unofficial solutions of the alkaloid differ very materially. There is no official solution ordered in the United States Pharmacopœia. The British Pharmacopœia has a solution of one per cent. and a hypodermic solution of ten per cent.

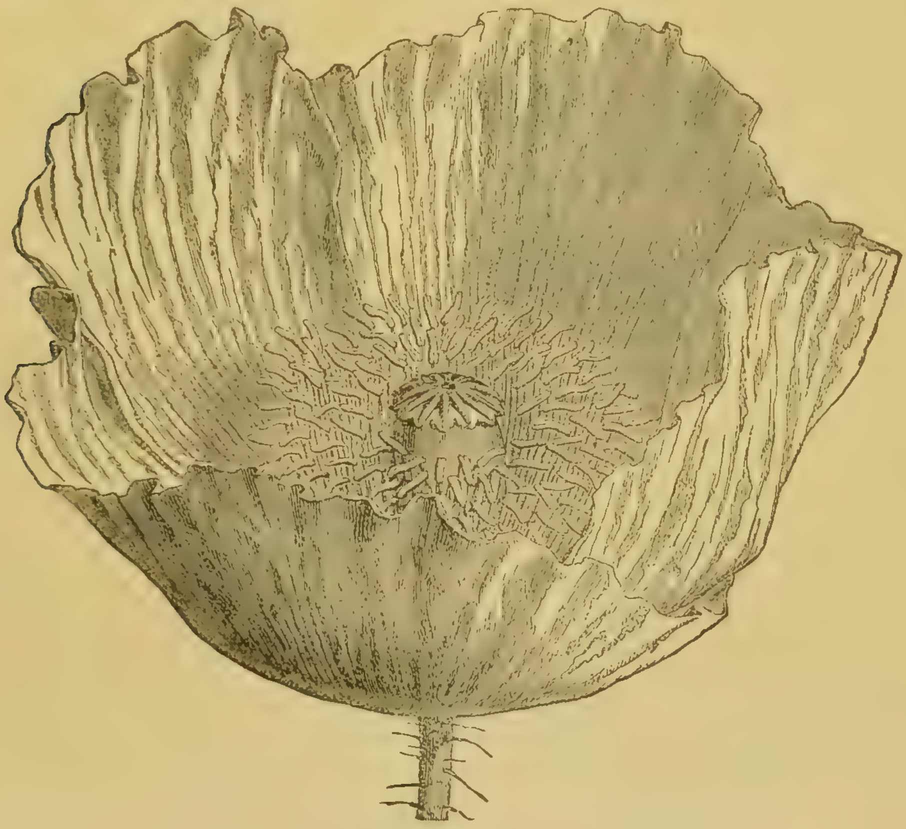

FIG. 36.—Flower of the White Poppy, Somewhat Reduced.

Morphine is not so rapidly nor so freely absorbed as atropine, strychnine, and many other alkaloids. When administered by the mouth its toxic effects are not detected for about fifteen minutes. If it has been given in the form of opium or its preparations, an hour or more may elapse before it influences the system. The alkaloid is absorbed from the mucous membrane of the vagina and intestines and requires twice the length of time to produce its effects. Opium is also absorbed from ulcers and wounds and

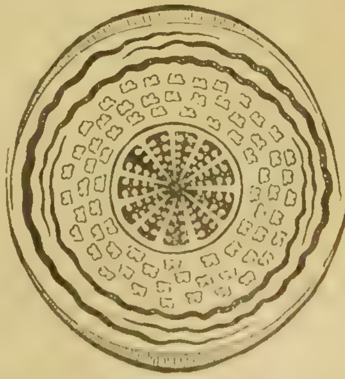

FIG. 37.—Plan of the Poppy Flower.

wherever the surface is denuded. It is doubtful if any absorption takes place through the unbroken skin. The poison is carried to all parts of the body and may be detected in any tissue. It does not exert any immediate change in the blood. The disposition of the alkaloid after being carried to the tissues is uncertain. A portion is probably decomposed, but the greater part is expelled unchanged. The urine and bile are its principal channels, but the skin and all secreting glands aid in its elimination. The probability of its excretion with the fluids of the stomach is now recognized and has become a point of practical importance, as the poison may be reabsorbed by the stomach or bowels and continue the toxic process. Its excretion begins early but proceeds slowly, the rapidity being retarded by the diminished functional activity of the various organs. The excretion is rarely completed before forty-eight hours and may continue for several days. The frequent repetition of an ordinary dose gives rise to an accumulation of the poison in the system. The repetition of small doses, however, only leads to a toleration of the drug.

Morphine exercises primarily a stimulating effect upon the nervous system, which is followed by one of depression, and in fatal cases death is due to paralysis of the respiratory nerves. The drug acts directly upon the nerve cells and influences the spinal cord, medulla, brain, and all parts of the nervous system. The early stimulation depends upon the size of the dose and the rapidity of its absorption. When the quantity is small, or is in such a form that absorption is slow, the stimulant action may be quite prolonged; if the absorption is rapid the early effects may be very transient, and depression then begins rapidly, or in some instances the stimulation may be supplanted altogether. The primary action causes an increased action of the heart and raises the blood pressure. The cerebrum is stimulated, causing an exaltation of the perceptive and intellectual faculties with which there is associated a varying degree of excitement. The beginning of depression is seen in the dulling of sensation, loss of reflexes, diminished muscular power, diminished secretions, irritation of the mucous membranes and itching of the skin, contracted pupils, vomiting, cerebral depression with drowsiness and tendency to sleep. The medulla is affected later and the lowered blood-pressure and weakened heart's action and respiration follow the other symptoms. In excessive doses there is complete loss of sensibility, coma is profound, respiration becomes slow and labored, and the heart's action is at first slow and weak, but toward the last increases in rapidity and feebleness. The skin becomes pallid and cold and covered with moisture, the features ghastly, the lips grow livid, circulation fails, and death takes place from asphyxia and respiratory failure.

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The *symptoms* of poisoning may be slow in appearing and gradually increase in severity, or the poison may suddenly overwhelm the system and the full effects of the drug be manifested at once. Ten or fifteen minutes may be sufficient to bring the system under its influence, or it may require an hour or more. In some cases four hours have elapsed (Taylor). The progress of the symptoms is comparatively slow and the duration of poisoning varies. Christison states that in fatal cases death takes place in from eighteen to twenty-four hours; Haines states they terminate in six to twelve hours. Cases have been reported in which death did not occur for fifty-six hours (*Medical Times and Gazette*, March 20th, 1858). In other cases it has taken place in forty-five minutes (Haines) and in one hour and twenty minutes. In some cases the symptoms may be severe and the prognosis exceedingly grave, and yet recovery take place. At other times the favorable progress of the case may be interrupted by an exacerbation of the symptoms which terminate in death. When the tendency is to recovery, an improvement is detected in the pulse, respiration, and color of the skin. The contraction of the pupil and the unconsciousness are less important signs, as they may be very persistent although a favorable course is being pursued. Many of the symptoms may continue as sequelæ and last for several days after recovery. Those generally noticed are contracted pupils and disordered vision, numbness and tingling of the extremities, and itching of the skin. Nausea, vomiting, constipation, and a deranged digestive tract are to be expected.

The fatal quantity is very variable. One grain of morphine, or its equivalent in opium, is generally considered as a dangerous dose and one liable to cause death. Von Boeck in the article on poisoning in Ziemssen's "Cyclopedia" says one grain and a half. Taylor has given two and a half grains of the extract, four grains of opium, and two drachms of laudanum as the smallest fatal doses. In other cases one drachm of laudanum has been sufficient to cause death in an adult (*Glasgow Medical Journal*, May, 1872). Some persons are very susceptible to the influence of opium and very minute doses cause all the symptoms of poison. One-third of a grain of morphine subcutaneously caused alarming symptoms (*Lancet*, September 30th, 1882) and one-sixth grain, in a case mentioned by Blyth, almost immediately caused unconsciousness, with livid face and almost imperceptible pulse. The aged and debilitated are most easily affected. Infants are peculiarly susceptible during the first year of life. This is very noticeable and for children up to five years of age it is considered a dangerous drug. Very small doses have proved fatal at this period of life. Taylor refers to a case in which

$\frac{1}{16}$ grain of opium was fatal, and in Ziemssen's "Cyclopedia" fatal cases are reported from one, two, three, and four drops of laudanum. Blyth considers $\frac{1}{10}$ grain of opium as a dangerous dose for children.

Recovery has followed the ingestion of very large quantities. Haines mentions two drachms of morphine, and in Ziemssen's "Cyclopedia" cases are reported of six and twenty-four grains of morphine having been taken and recovery followed. Such results are to be accounted for by the variations in strength of the different preparations of the alkaloid, the slowness of action of the drug, and the frequency of vomiting.

A man, 28 years of age, took an unknown quantity of laudanum. He became drowsy; the stomach was washed, hot coffee was injected, and then he was "walked about." Afterward, however, he became worse, pupils contracted, pulse slow, full; he became comatose; $\frac{1}{2}$ grain atropine was administered subcutaneously. In less than five minutes the pupils became dilated and he sat up and said he felt all right; in half an hour he became suddenly comatose again; the breathing ceased, but the pulse continued good. Artificial respiration had to be kept up for three hours until the respiratory movements were re-established; he became conscious the next morning (*Lancet*, 1894, II., p. 128).

Girl, 19 years old; received about 12 grains of morphine hypodermically. In a few minutes she was in a stupor, with pupils contracted. The treatment consisted in galvanism, slapping with wet towels, coffee, exercise, artificial respiration, and atropine, 4 grains in $\frac{1}{20}$-grain doses, during twelve hours. She became very comatose, the respirations ceasing at times; the pupils were not dilated; for twelve hours she remained comatose and required treatment for thirty-six hours. She finally recovered (*Therapeutic Gazette*, September, 1894).

Adult, male; took an unknown quantity hypodermically. When seen, he was insensible, pupils contracted; respirations 4 to 6; pulse 60 per minute. One-fiftieth grain atropine sulphate was given hypodermically with the result that the pupils dilated and the pulse increased. Three hypodermics of the antidote were given, which improved the pulse and dilated the pupils, but exerted no influence on the respiration. Artificial respiration was maintained for several hours. Recovery followed (*Therapeutic Gazette*, 1893, S. 39).

A man, 36 years old, drank a quantity of laudanum equal to 17 grains of opium. In twenty minutes he was comatose; pupils contracted, pulse good; respiration weak and failing. The treatment consisted in artificial respiration, the use of the stomach-pump; brandy, coffee, and exercise. Artificial respiration was resorted to several times. After three hours $\frac{1}{60}$ grain atropine was given and repeated in one-half hour. Respiration increased in frequency and improved. No further artificial respiration was required; in about six hours improvement began and continued steadily for forty-eight hours (*Lancet*, 1884, II., p. 429).

Infant, 18 months old, ate two $\frac{1}{4}$-grain tablets; it became unconscious; the pupils were contracted; in a short time 1 grain permanganate of potassium was introduced into the stomach and three hours later another grain. No emetics or any other remedies were used. The following day the child had quite recovered (*Medical Record*, November 5th, 1894).

A man, 32 years of age, took 8 grains of morphine; in three hours he was unconscious, pupils contracted; respirations 15 per minute and stertorous. The treatment consisted in flagellation, cold affusions, interrupted current, ammonia, and ether, used without effect for one-half hour; respiration reduced to eight per minute, pulse almost imperceptible. Inhalation of pure oxygen was begun; in twenty minutes the respiration, pulse, and general appearances improved. In one-half hour the inhalations were repeated and in one-half hour they were given for the third time, when consciousness returned. During the day the man became drowsy but there was no coma (*Ontario Medical Journal*, September, 1894).

In the *treatment* of all cases of opium poisoning attention must be given to the evacuation of the stomach and the removal of any portion of the poison that may remain unabsorbed. The comparative slowness of its absorption makes this of the utmost importance. Emesis is to be attempted, and is particularly desirable if the crude opium or one of the solid preparations have been taken or if food remains in the stomach. The stomach-pump should always be employed whether the emetics have acted or not. A thorough washing is to be made and every care taken to remove every trace of the poison. At the same time tannic acid, or any vegetable astringent, is to be added for the purpose of precipitating the alkaloid and rendering it less soluble. Animal charcoal may also be given for the same purpose. This should not be added to the water but given in the dry state or in rice paper. Before removing the stomach-tube, it is recommended to inject a quantity of hot strong coffee and allow it to remain in the stomach.

For many years it has been known that after subcutaneous injections the normal secretions of the stomach contain the alkaloid, and that a certain proportion of the poison is removed from the system in this way. The same excretion takes place after the poison has been taken into the stomach and absorbed into the system, and this is now taken advantage of, and the possibility of a reabsorption and re-poisoning averted by repeatedly washing out the stomach and removing the poison as it is secreted into this viscus. It has been estimated that nearly one-half of what has been administered hypodermically may be recovered from the secretion of the stomach.

In experimental work it has been demonstrated that ten or twelve centigrammes per kilogramme is the fatal quantity for dogs. When lavage is performed this fails to produce death and much larger doses can be given without fatal results. In the *Johns Hopkins Hospital Bulletin*, No. 94, 1894, a case is fully reported in which the poison was taken at ten o'clock in the morning, the stomach was cleansed at five o'clock, and again at eight and at eleven o'clock in the evening, and each time the presence of morphine was clearly demonstrated.

Upon the same grounds the urine should be removed to prevent any possibility of reabsorption by the walls of the bladder. The bowels should also be freely evacuated.

The elimination of the poison will be assisted by spirit of nitrous ether and copious draughts of water. Pilocarpine has also been employed for the same purpose.

Coma is to be combated if possible by arousing the patient from his drowsiness, by means of flagellation and constant movement. The fact that the muscular power remains until the influence of the poison is far advanced allows of this being readily carried on; but when there is much prostration and feebleness care must be exercised not to carry enforced movement to excess and so exhaust the patient.

Stimulants will be indicated where the respiration and circulation are failing. The hypodermic use of brandy, ether, and camphor is serviceable. Lately strychnine and caffeine have been recommended; and very many cases are reported in which they have been used with marked benefit.

In addition to the above measures attempts must be made to neutralize the effects of the poison after absorption, and atropine is very generally selected for this purpose. This drug has been in use since the sixteenth century. Its early employment was based upon its well-known antagonistic action upon the pupil, and later researches seemed to indicate that it had a stimulating action upon the heart and respiration. From its earliest use, however, its value as an antidote has been questioned, and recent investigations have failed to detect any stimulant effect upon the respiration (*Archives of Pediatrics*, April 19th, 1893; *Texas Medical Journal*, December, 1892; *Universal Medical Journal*, February, 1891). Many utterly condemn the employment of one drug as an antidote to the other, claiming that the coma and depression are increased and a double poisoning caused. It was formerly directed to administer atropine until the dilatation of the pupil was established and maintained. It is now advised to be given in doses of $\frac{1}{20}$ grain repeated in fifteen minutes, not to be administered more than three times. Notwithstanding the evidence of experimental work against the value of atropine as a respiratory stimulant, the numerous cases in which this method has been used with apparent benefit will keep it prominent as a means of treating these cases of poisoning.

Permanganate of potassium is the latest addition to the list of antidotes (Moor, *Medical Record*, February 17th, 1893; Chambers, *Canadian Practitioner*, December, 1894; Wood, *Universal Medical Magazine*, August, 1894; *Medical News*, May 12th, 1894). Dr. Moor found that when this was brought in contact with morphine it decomposed the alkaloid and rendered it harmless. He demonstrated its properties by experiments and by partaking of large doses of the poison and its antidote. One grain neutralizes an equal weight of the alkaloid, but on account of the organic matter present in the stomach an excess of the antidote is required. A one-per-cent. solution is recommended which may be used freely, as in this strength it is harmless. When the poison has been administered hypodermically, the question of its excretion by the gastric mucous membrane is to be considered, and the permanganate is then recommended for the purpose of neutralizing it as excreted into the stomach. Much clinical and experimental evidence of its value has been forthcoming and it is very generally looked upon as a successful means of treating this poison. In many of the reports of poisoning treated with the permanganate, it has been introduced into the system subcutaneously, in some instances without any attempts to attack the poison in the stomach. To what extent this may prove of value is uncertain, as its action upon the blood is not understood and may prove to be highly injurious. Experiments by Wood indicate that when administered subcutaneously the antidote had no effect upon the poison, but apparently hastened the death of the animal experimented upon.

Whatever doubt there may be regarding the value of therapeutic antidotes there can be no question as to the importance of artificial respiration. When the coma has become profound and the respiration begins to succumb to the influence of the poison, the effort to maintain life must be supplemented by artificial means. In some instances, where the respiratory movement is variable, a few movements at the critical period will avert death; in others it may be necessary to continue steadily for several hours until the system has excreted the poison and recovered from its effects.

Inhalations of oxygen have also been recommended and in many instances have proved of much service. In addition to its usefulness in overcoming the cyanosis and stimulating the circulation, it is also supposed to exert a direct oxidizing effect upon the poison. This idea has been suggested by the similar property of the permanganate of potassium, but as early as 1860 Dr. B. W. Richardson advocated the employment of hydrogen peroxide for the same purpose on account of the oxygen it contained. He directed it to be used subcutaneously, and by transfusion directly into the circulation, as well as by vaporizing it and inhaling.

Another almost indispensable adjunct in critical cases is the application of electricity. Both the faradic and galvanic currents have been used with success, in some instances being persisted in for several hours.

The post-mortem appearances furnish nothing upon which to base an opinion as to the cause of death, except in the cases in which the contents of the stomach furnish evidence of the presence of the poison. The only condition that is of any importance is the congested state of the vessels of the brain and its membranes, and the frequency of effusions into the ventricles. This, however, is not characteristic and may not be very evident. The blood and organs are in such a state as is common to asphyxia.

The poison may be detected after death in any of its tissues and secretions. Of the former the liver, kidneys, stomach, and blood are generally selected. The secretions most likely to contain it are the urine, bile, fæces, and contents of the stomach. It has been detected many months after death; Tidy gives an instance of four months, Taylor of fourteen months. The presence of ptomaines simulating the reactions of morphine may lead to mistakes (see Vaughan, "Ptomaines and Leucomaines").

CODEINE, $C_{18}H_{21}NO_3$, and its official salts, the sulphate and phosphate, are coming into very general use and have occasionally been the cause of poisoning. The percentage in opium is very small compared with morphine, ranging from .2 to .75 per cent. It is obtained in white octahedral crystals, soluble in eighty parts of cold and in seventeen of boiling water. It is freely soluble in alcohol, ether, chloroform, and solution of ammonia. It is alkaline in reaction and combines with acids to form much more soluble salts. Chemically codeine is considered the methyl-ether of morphine, and it has been prepared from the latter alkaloid (*Journal of the Chemical Society*, 1881, 829).

Codeine exercises the same action as morphine upon the cerebrum but in a less degree, and, in addition, has a special stimulant action upon the spinal centres. It produces sleep, which is less restful and profound than that of morphine, it contracts the pupil, causes irritation of the skin, and has the same depressant effect upon the respiration and heart. Its action upon the cord is manifested in the greater degree of restlessness and excitability, twitching of the muscles, spasms, and convulsions. Vomiting is more frequent and purging often replaces the constipation produced by morphine. Codeine is five or six times weaker than morphine, the dose being from one to two grains. What quantity is likely to be dangerous is uncertain, as instances of poisoning are not common. Falck (Blyth), as a result of experiments, has determined that 51.2 milligrammes per kilogramme is the fatal dose for rabbits. As with morphine, the system becomes accustomed to the drug when frequently administered, and very large doses may then be given with impunity.

A woman, who had been taking $\frac{1}{4}$-grain pills for some painful disease, one night took sixteen $\frac{1}{2}$-grain pills; this caused nausea and vomiting. When seen in three hours she was restless and irritable but able to converse, kept tossing about the bed and constantly changing her position. At frequent intervals she had convulsive movements involving the entire voluntary muscular system, marked hyperæsthesia of the skin and intense irritation along the flexor surfaces of the arms, and on the back. The skin was dry; the pupils contracted; respirations 12 per minute. Complained of fulness in the head and thirst. For some time she would occasionally sink into a light doze, from which she would awake with a start. She was given the "usual treatment for opium-poisoning" and rapidly improved. The following day she had entirely recovered (*Therapeutic Gazette*, 1894, 32).

Adult took 4 grains of codeine. This dose caused excitement, restlessness, delirium, followed by vomiting, depression, contracted pupil, pallor of the surface, and cold perspiration (*British Medical Journal*, 1874, I., 478).

The *treatment* adopted for these cases of poisoning is that for poisoning by opium and morphine. The irritability of the stomach facilitates the action of emetics and the poison is readily removed. Purgatives and diuretics are indicated, and a free action of the skin should be secured. Atropine will prove useful in lessening the cerebral irritability and in securing quiet and rest as well as benefiting the depressed respiration. In advanced stages the usual methods of combating the vital depression and tendency to asphyxia will have to be employed.

Solanum.

The various species of the genus Solanum, order Solanaceæ, are possessed of poisonous properties, somewhat similar to belladonna, hyoscyamus, stramonium, and other genera of this order. The species are widely scattered, the most poisonous forms growing in the hotter climates of South America and Asia. Those found in America are *Solanum dulcamara*, L., "Bittersweet"; *Solanum nigrum*, L., "Nightshade"; and *Solanum carolinense*, L., "Horse-nettle";—all of which are comparatively common throughout the United States and Canada. *Solanum tuberosum*, the common potato plant; *Sola-*

num lycopersicum, the tomato plant; and *Solanum indigoferum*, from which indigo is obtained, are also members of this family and contain the same active principle.

S. dulcamara is a perennial vine, with a weak, half-woody stem, growing to a great length; dull green alternate leaves, heart-shaped below and tapering above. The flowers are in spreading, extra-axillary cymes, purplish, star-shaped, about half an inch across. The fruit is an oval shining berry, light green at first and becoming yellow, orange, and scarlet as it ripens. The young branches were formerly official. The taste is bitter at first, becoming sweet afterward.

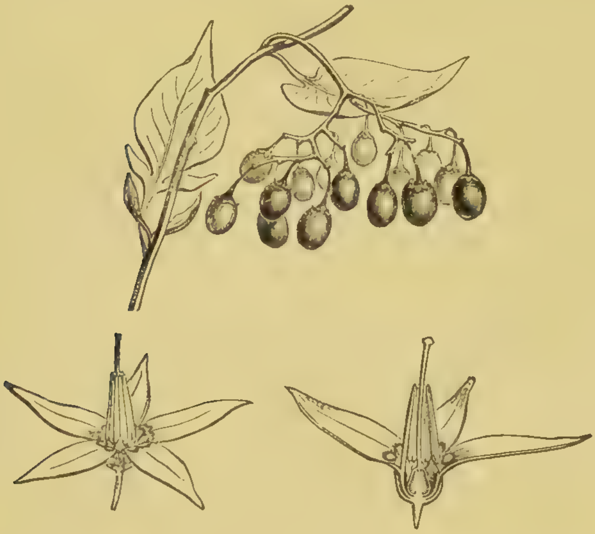

FIG. 38.—Bittersweet. Plant reduced and flower enlarged. (Baillon.)

S. nigrum is a low annual, with numerous spreading and branching stems; leaves oval and toothed; flowers white and small; the fruit is a greenish-black berry. The whole herb was formerly official.

S. carolinense is a low, hardy perennial, about one foot high; stem is erect, prickly, leaves ovate, rough, and pubescent and prickly; flowers are pale blue or white; fruit globular, orange-yellow in color.

The properties of the solanums depend upon the presence of an active principle, *solanine*, $C_{42}H_{87}NO_{16}$, which exists in variable amounts in the different species and in many is only present in certain parts of the plant and at certain periods of its growth. It is most abundant in the young stems and fruit. The active principle is generally considered an alkaloid, but this has been questioned, as the action of weak acids decomposes it into dextrose and solanidin. It is obtained as colorless, glittering crystals, resembling mother-of-pearl in appearance; its taste is extremely bitter. Its aqueous solution froths upon shaking. Solanine is readily obtained from the young shoots of the potato.

The action of solanine resembles that of atropine in many respects, but is not very well understood, the reports of its effects being very contradictory. It is supposed to depress the motor centres, causing the heart and respiration to become weak and feeble, and impairing the muscular power. Sensibility is also diminished. The respirations are rapid at first, later they become slow and irregular; the pulse also becomes irregular and intermittent. There is general apathy, indisposition to voluntary movements, convulsions, and coma before death. Various statements are made regarding its mydriatic powers. It is supposed that the pupils are contracted by solanidin, but not by solanine. Solanine is recommended as a substitute for morphine in the treatment of neuralgias, and in bronchial and asthmatic affections. All parts of the plants have been the cause of many poisoning accidents, the bright attractive berries being most frequently the source of the poison. The bittersweet berries gave rise to alarming symptoms in a child eleven years of age (*Pharmaceutical Journal*, 1861, 436), and a child, fourteen years old, died on the second day after having eaten some green potato fruit (*British Medical Journal*, September 3d, 1859).

Solanine exists in the fruit of the bittersweet in the proportion of about $\frac{1}{3}$ of one per cent.; the dose is recommended to be $\frac{1}{4}$ to 1 grain. The fatal quantity is not determined. Blyth states that about one and a half grains per kilogramme is fatal to rabbits.

The *treatment* consists in removing from the stomach, by the aid of emetics, such portions of the poison as have not been expelled by vomiting induced by the poison itself; purgatives also should be given to expel such portions as may remain in the bowels. The general treatment must be the same as that for poisoning by atropine, stimulants if the depression is profound, and opium to relieve the pain and quiet the restlessness and irritability.

Stramonium.

Datura stramonium, L., Thorn-apple, Jamestown or "jimson" weed, order Solanaceæ, is a common plant throughout America and all other temperate and warm countries. It is found in waste places and cultivated fields, in and about towns and villages. It is a coarse fetid weed, growing from one to six feet high. The stem is green, hollow, and branched; the leaves are petioled, ovate, sinuate-toothed with wavy margins; the flowers are solitary, springing from the axils of the leaves with short peduncles; the corolla is white, funnel-shaped, convolute, the border with fine teeth; the fruit is a four-celled pod, the size of a small

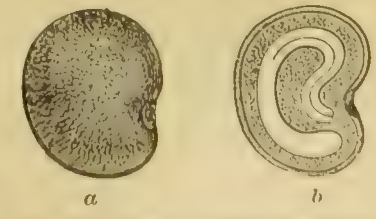

Fig. 39.—Seed of Datura Stramonium *a*, seed entire; *b*, the same in longitudinal section showing the embryo.

egg and covered with spines; the seeds are numerous, brownish-black. The whole plant has a disagreeable odor, particularly noticeable when bruised, but lost upon drying. *Datura tatula*, L., is another species which is not so common in this country. Its stem and flowers are purplish. Other species which are much more virulent occur in Central America and India.

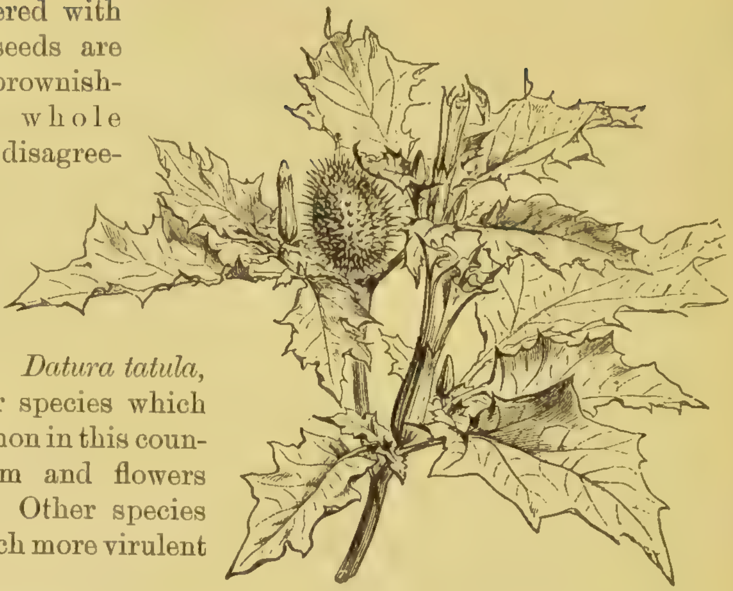

FIG. 40.—Datura Stramonium. Branch, showing flower and fruit.

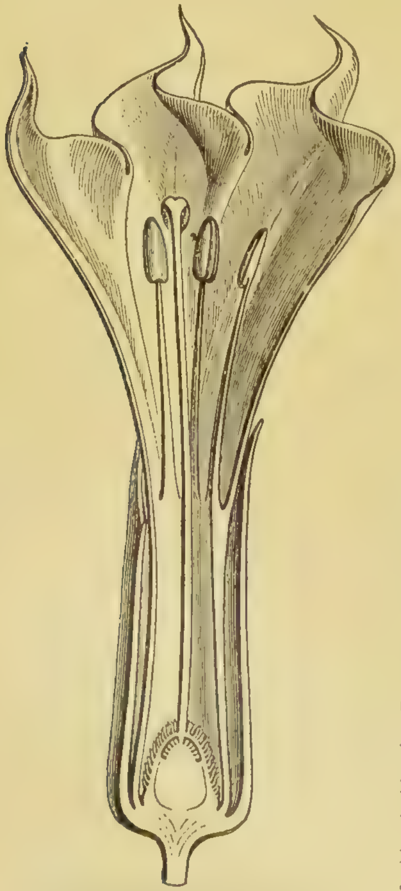

FIG. 41.—Flower of Datura Stramonium, in Longitudinal Section.

The properties of the plant depend upon the presence of *daturine*, an alkaloid now considered to be identical with atropine and hyoscyamine. It exists to the extent of one-fourth of one per cent. in the seeds. The leaves are about one-third as strong as the seeds.

The *symptoms* of poisoning are very similar to those of belladonna poisoning, but stramonium exerts a more marked depressing effect and diminishes sensibility to a greater extent. Its action upon the respiratory organs and heart is very decided. The muscular system is relaxed and diarrhœa and profuse perspiration may occur. The cerebral symptoms are more severe than in atropine poisoning and maniacal excitement may take place with delirium. The pupils are dilated in less degree, the throat is at first dry, and the skin is dry and hot; convulsions and palsy may take place.

A woman, aged 30 years, took, before going to bed, one ounce of infusion of the leaves, 1 part in 16. Shortly after falling asleep she was noticed to be restless and breathing deeply and loudly, and on attempting to rouse her she appeared to be out of her mind. Pulse 130, thready; respirations shallow, 35; conjunctivæ deeply injected, eyes wildly staring and restless. Face and chest were covered with erythema. Tongue and fauces were dry. Delirium became wild and active with hallucinations of sight and hearing. Patient grew almost unmanageable, breaking everything within reach and constantly looking under the bed and searching for strange men and animals. Half a grain of morphine was given hypodermically and in one hour she was resting. The next morning all symptoms had disappeared (*Medical Record*, October 21st, 1893).

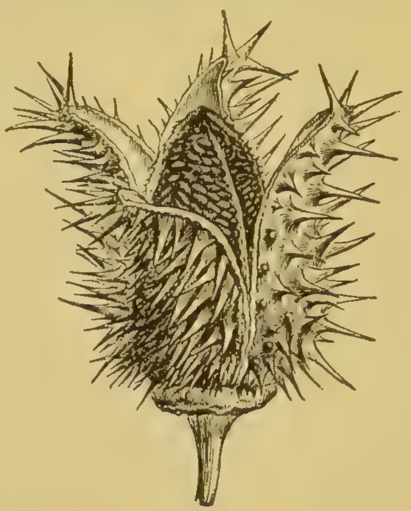
FIG. 42.—Ripe Fruit of Datura Stramonium.

The indications for *treatment* are the same as in cases of poisoning by belladonna.

Strychnine.

Strychnos is a genus of the order Loganaceæ containing numerous species, of which the best known are *Strychnos Nux Vomica* and *Strychnos Ignatii*, both having *strychnine* and *brucine* as their active principles. The latter species is the more powerful, as it contains nearly twice as much strychnine as the first named. *Strychnos tieuté*, the upas tree, from which the Java arrow poison is obtained, and *Strychnos toxifera*, the chief source of curare, also belong to the same genus.

Strychnos Nux Vomica is a medium-sized tree; with a short, and frequently crooked stem; irregular branches, without spines or tendrils. Leaves are opposite, thick, short-petioled, broadly oval, bluntly pointed at apex and base, bright green and shining above, smooth. Flowers are perfect, in terminal corymbs of a greenish-white color. Calyx short, fine-toothed. Corolla funnel-shaped. The fruit is a large, round, yellow berry, covered with a smooth, hard shell and filled with a white, soft, gelatinous pulp, in which are several seeds. The seed is about one inch in diameter, round and flat, concave on the ventral surface, and flat or slightly convex on the other. On the ventral surface is the remains of the umbilicus. The testa is covered with a layer of short silky hairs, the substance of the seed is hardened albumin, of a dirty white color. At its margin is situated the embryo.

All parts of the plant contain the alkaloids, but the seeds only are employed in medicine and are familiar to us as nux vomica. Several years ago the bark was introduced into commerce under the name of false Angostura bark and was the cause of many severe cases of poisoning.

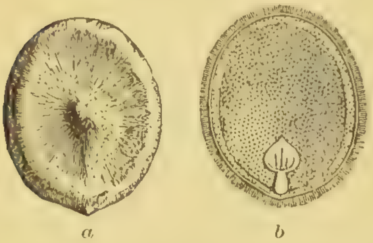

FIG. 43.—Seed of Strychnos Nux Vomica—*a*, seed entire; *b*, section of seed, showing the embryo.

Nux vomica is met with as the whole seed and in powder. The latter has occasionally been the cause of some accidents, but it is the alkaloid that is nearly always the form from which the poisoning occurs.

The alkaloids are *strychnine* and *brucine*, which exist in combination with *igasuric* or *strychnic acid*. A third alkaloid, *igasurine*, has been described but it exists in small amounts and has not been definitely studied. The physiological action of all is similar, varying only in intensity.

Strychnine, $C_{21}H_{22}N_2O_2$, exists to the extent of about one-half to three-fifths of one per cent. It occurs as small, colorless crystals, odorless, but of an intensely bitter taste which is imparted to water, 1 part in 70,000 being readily detected. Haines (Hamilton's "System of Legal Medicine") states that some persons are unable to taste any bitterness in solutions of a much greater strength, a fact of some medico-legal importance. It is not very soluble, requiring 6,667 parts of cold and 2,500 of boiling water. It is readily soluble in boiling alcohol but separates from it when cold, as it requires 200 parts to retain it in solution. This allows it to be separated from brucine. It is alkaline in reaction and combines with acids to form salts, which resemble it in appearance but are much more soluble. The salts are about one-fourth weaker than the alkaloid.

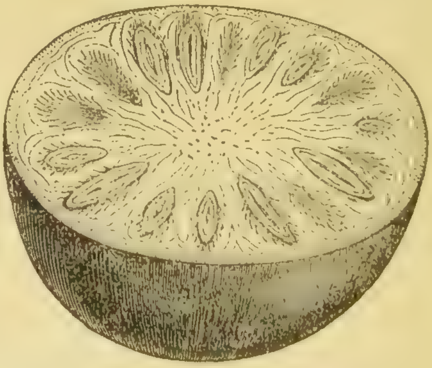

FIG. 44.—Transverse Section of the Fruit of Strychnos Nux Vomica. The seeds are here more regularly arranged and more numerous than in most specimens.

Brucine, $C_{23}H_{26}N_2O_4$, resembles strychnine in appearance, occurring in colorless prisms, or in white flakes due to efflorescence. It dissolves in 320 parts of cold and 150 of boiling water, the non-crystalline form being less soluble. It is very soluble in both hot and cold alcohol. Its taste is exceedingly bitter, and it combines with acids to form salts. The porportion of brucine in nux vomica is greater than that of strychnine, ranging from one to two per cent. Its physiological action is the same, but it is estimated to be thirty-eight times weaker and much slower in producing its effects. Richert (*Medical News*, April 8th, 1893) publishes a series of experiments to determine the comparative actions of the two alkaloids. He finds no difference of any practical importance, excepting in their strength. He considers strychnine forty or fifty times as powerful as brucine. Brucine and strychnine may be separated by their relative solubility in alcohol, but this is more thoroughly accomplished by adding a neutral solution of chromate of potassium to a hot solution of the salts, chromate of strychnine being deposited as insoluble, orange-yellow

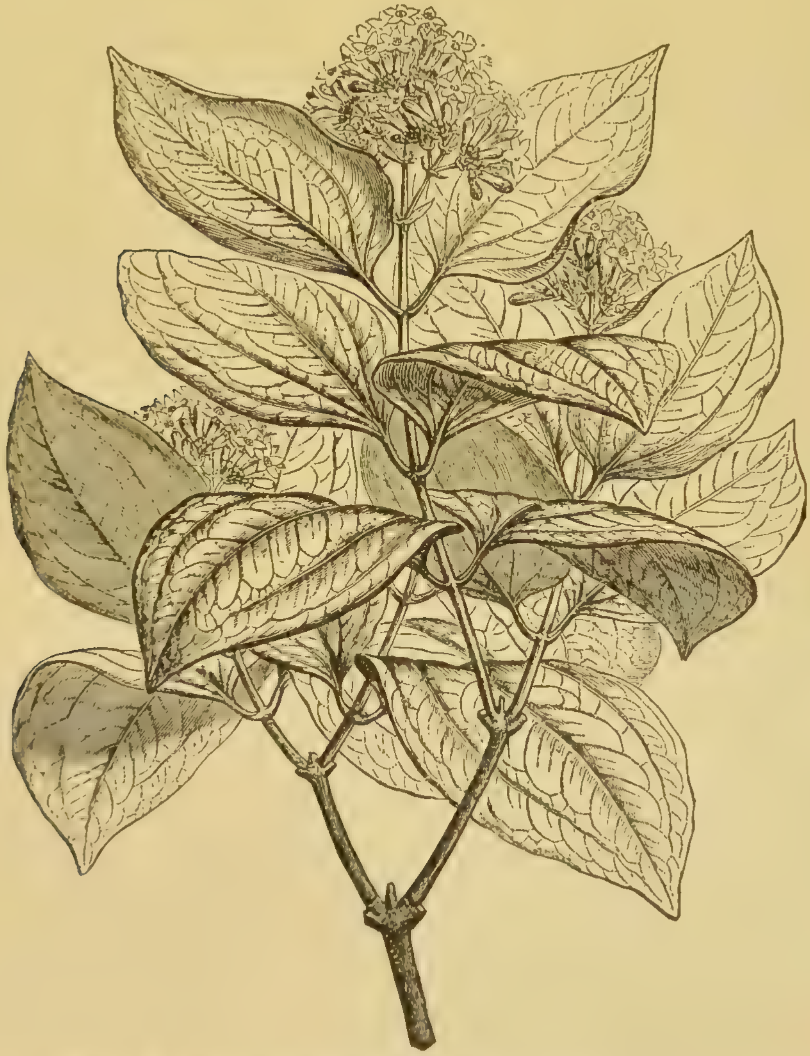

FIG. 45.—Strychnos Nux Vomica, Flowering Branch. (Baillon.)

crystals. As a test for the presence of brucine, Blyth states that its reaction with methyl iodide is the most reliable. To a solution in strong alcohol a little methyl iodide is added, and in a short time circular growths of crystal groups appear, which are composed of methyl brucine iodide. Upon treating brucine with nitric acid a characteristic color reaction is produced; a bright red color appears which upon warming fades to a yellow, and upon the addition of a solution of stannous chloride a bright purple coloration takes place. This latter part of the test is the distinctive reaction.

Strychnine is a motor irritant, its effects being directed to the motor centres of the spinal cord, increasing their irritability in a very marked degree. When this hyper-sensitiveness is maintained for a prolonged period, or when an excessive effect is produced, there follows a secondary state of depression or paralysis of the same centres. That the effect is spinal in origin has been proved by separating the brain from the cord without lessening the reflex action. It has also been shown that the irritability is not peripheral by dividing the nerves to the part. The sensory as well as the motor nerves are not affected and the brain remains uninfluenced. The muscular tissue is also free from any direct effect of the poison. Its action upon the blood is uncertain, but its oxygenation is interfered with, and this direct action, in addition to the spasm of the respiratory muscles and the increased tissue metamorphosis, favors the accumulation of carbonic acid in the system and in time tends to cause asphyxia.

Richert (*Therapeutic Gazette*, 1892) has published a series of experiments which demonstrate very clearly the excitant and paralyzing action of strychnine and show in detail the effects of the poison upon the various functions. His experiments were based upon the discovery of Richter and others (Ziemssen) that when artificial respiration is performed the effects of poisonous doses may be overcome. He prefaces his work with the following résumé:

"When a one-per-cent. solution of sulphate of strychnine is injected into the external jugular vein of a dog there occurs within a few moments a condition of muscular excitement, which rapidly passes to one of tetanic convulsions, and death ensues immediately from an arrest of respiration, or, after a time, from exhaustion. Should, however, forced artificial respiration be maintained at the time of the convulsive seizures and after the spasms gradually disappear, the general condition of the animal becomes in most respects the same as just before the convulsions, and the effects of the poison slowly wear away. But if with the appearance of the convulsions the injection of the strychnine is continued, the tetanus grows less and less violent, voluntary motion is lost, reflex excitability rapidly disappears, the spasms give place to somewhat rhythmical jerkings of the muscles, and these to twitchings, which in turn are lost, and the animal lies absolutely motionless, save for the movements due to artificial respiration and the heart-beats. The circulation, heat-processes, and other important functions remain in good condition, and the dog may be kept alive for hours, if not for days, thus paralyzed, if strychnine is from time to time injected. These remarkable changes from the normal state to violent convulsions, and finally to complete motor paralysis, occur with such rapidity that the latter stage may be present within three minutes."

The *symptoms* of strychnine poisoning are very characteristic and any mistake in diagnosis is due to a lack of judgment on the part of the observer rather than to any obscureness of the symptoms. There is at first a sense of uneasiness and sensitiveness, a tightness of the chest, a "catch" in the respiration, and an occasional twitching and stiffening of the muscles. There may also be heat or a pain in the epigastrium, and sometimes vomiting. These premonitory symptoms are followed by a sudden onset of tetanic spasms of a more or less violent character. The muscles of the extremities are first affected, but the spasms rapidly extend to the muscles of the body and increase in severity until opisthotonos is produced. The chest-walls also become fixed and respiration is checked during the spasms. The muscles of the face are early affected causing curious distortions, but the trismus is not so early nor so marked as in tetanus. The impeded circulation gives rise to fulness of the veins, congestion of the face, protruding eyeballs, and in later stages to all the distressing signs of impending asphyxia. The seizures vary in duration from a few seconds to several minutes. They recur more or less rapidly, in some instances within one minute, in others there is an interval of more than an hour. Gentle and continuous movements or sounds may not affect the patient, but any sudden, startling impression, however slight, may be instantly followed by a severe paroxysm. Death usually takes place during a seizure, and when these are severe life may terminate in the third or even in the second attack. When death takes place during the interval it is accounted for by exhaustion and paralysis of the spinal and medullary centres. When the quantity of poison is excessive the nervous system appears to be overwhelmed, and death may occur almost instantly without any convulsive attack.

The alkaloid is rapidly absorbed and the onset of symptoms is rapid, but the form in which the poison is administered may delay its absorption. When injected subcutaneously symptoms of poisoning may follow almost immediately. In experiments upon dogs tetanus has been produced in twelve seconds. Witthaus ("Reference Handbook of the Medical Sciences," Vol. VI.) gives a table of 49 cases in which the symptoms appeared "directly" or "very soon" in 12, in from three to thirty minutes in 22, in from thirty minutes to one hour in 7, in one to two hours in 6, and in two and one-half to three hours in 2. When the poison is given in pill form or as dry and old extract, the solution may be slow, and long intervals may thus elapse before symptoms appear. The powdered nux vomica is slow in its action compared with the alkaloid, half an hour being given as the time required for its effects to begin. The presence of food will retard and the ingestion of fluids hasten the onset. The course of the symptoms is rapid and in fatal cases death may occur very quickly. Of 40 fatal cases collected by Witthaus, 20 died within one hour, 8 in from one to two hours, 7 in from two to three hours, and 5 in from five to six and a half hours. In 21 cases the time between the onset of symptoms and death was within half an hour in 8, from a half to one hour in 6, from one to two hours in 4, and from two to five hours in 3. Death is frequently recorded as taking place "immediately" and cases are reported in which only two and three minutes have elapsed. When recovery is the result the course is also rapid. Ringer states that if the patient survives for two hours a favorable termination is to be expected. The prognosis, however, must be guarded, as the symptoms may last six and eight hours before death takes place. In some instances the condition has suddenly become worse when the case was apparently progressing toward recovery.

The excretion begins early and the poison has been detected in the urine within half an hour. Its excretion, however, is not so rapid as its absorption and the poison thus accumulates in the system. It is supposed to be wholly excreted from the body in two days.

A large percentage of the cases end fatally and very small doses may cause death. One-fourth of a grain proved fatal to an adult (*Medical Times and Gazette*, 1854, 376), also one-half grain (*British Medical Journal*, August, 1847). Taylor mentions an instance of death in a child from $\frac{1}{16}$ of a grain. Richert's experiments show that the smallest lethal dose for a dog is $\frac{1}{300}$ grain per 2.7 pounds (kilogramme) of body-weight. This would equal in a man, averaging one hundred and fifty pounds, about $\frac{7}{10}$ of a grain. Taylor mentions one-half grain as the smallest fatal dose, and Guy and Ferrier, one-quarter grain. Although such small amounts may cause death, very much larger quantities have been recovered from, ten to fifteen grains (*American Journal of the Medical Sciences*, 1856, 547), ten grains (*Lancet*, XI., 1856, 695), twenty grains (*British Medical Journal*, December, 1870). Many of such recoveries are traceable to prompt and energetic treatment, on account of which the amount absorbed is but a small proportion of that taken. Adulteration of the drug may also have some influence. In other cases suicidal attempts have failed owing to other drugs being combined which are antidotal in their action. Opium is very commonly taken with strychnine, and in one instance chloroform was inhaled at the same time that the poison was swallowed (*Lancet*, 1882, May 13th, November 28th).

The fatal quantity of the powdered nux vomica is from one-half to one ounce. Death has taken place in two hours after taking five drachms (Pellarin, quoted in Ziemssen's "Cyclopedia of the Practice of Medicine," American Edition, Vol. XVII., p. 780).

Tetanus is the one disease that may be mistaken for strychnine poisoning, and there may be much difficulty in arriving at a diagnosis. The history of the case, the high temperature, and early lockjaw, are generally well marked in tetanus; while the sudden onset, the rapid course, the commencement of the spasms in the lower extremities, and the late appearance of the lockjaw, in the case of strychnine poisoning are generally sufficiently clear to distinguish the two. Epilepsy has been mistaken for poisoning, and Blyth relates a fatal case in which hysteria was diagnosed and death assigned to that cause. Some forms of brain and spinal disease, and meningitis may resemble cases of poisoning. The diagnosis may also be made difficult by the combination of laudanum or other poisons with strychnine.

A man, 46 years old, took probably 7 or 10 grains strychnine. In three-quarters of an hour he was found in convulsions, most marked in lower extremities; chloral 40 grains, and bromide 1 drachm, were given per rectum, and the stomach pump was used. Paroxysms recurred every ten to twenty minutes, lasting from ten to twenty seconds. At times they became more severe but appeared to be controlled by chloral. At the termination of eight and three-quarter hours a sudden severe attack took place which ended fatally (*Australian Medical Gazette*, March, 1893; *Therapeutic Gazette*, July, 1893).

A man took about 3 grains of strychnine. In four or five minutes he felt ill. Cramps appeared in the legs and ascended to the thighs. While rubbing he suddenly fell, shrieking, in a spasm, which recurred frequently. In one hour and a half he died, while being lifted, from exhaustion; there was no convulsion at the time. The face was livid, lips blue and shrivelled, pupils dilated, and the muscles of the whole body relaxed. The man received no treatment. Post-mortem ten hours after death. Rigor mortis pronounced, abdominal and pectoral muscles in a state of tension. No priapism. Blood very dark and fluid, right side of the heart full, left side empty, all organs slightly congested. Meninges of the brain and cord engorged, also vessels of the brain and spinal cord (*Lancet*, November 9th, 1889).

A man took about 6 (?) grains of strychnine. In twenty minutes he was seen to be in tetanic spasm, with opisthotonos, arms flexed across the chest, rigid tremor, hands clenched, legs extended. The face was livid and congested, the features fixed in a spasmodic grin. At the onset of the spasm he gave a shriek; the attack lasted two minutes, with dilated pupils. The mind was clear in the intervals, the muscles twitched continuously at intervals of about three minutes; asphyxia threatened during paroxysms. Chloral, 20 grains, was administered subcutaneously and repeated in doses of 20, 10, and 20 grains each. Attack began to grow milder in half an hour, ceased in four hours, but twitchings continued for four or five hours. The man finally recovered (*Lancet*, July 27th, 1889).

A man, 56 years old; took 20 grains of sulphate of strychnine in less than ten minutes. Emetics were given, the stomach washed, and tannic acid, bromide of potassium, and chloral administered. Patient escaped with only twitchings of the muscles of the limbs and jaw and contractions of the œsophagus (*British Medical Journal*, August 21st, 1892).

Treatment must be prompt and energetic. The many instances in which large doses have failed to cause death, the facility with which, in experimental work, the lives of animals are preserved, and the effectiveness of drugs in overcoming the action of the poison should encourage one in the management of such cases. The first step should be the administration of antidotes to hinder the absorption of the alkaloid, and the removal of the contents of the stomach. Tannic acid and vegetable astringents precipitate the alkaloid and may be given freely. Iodine also causes an insoluble salt (*Lancet*, April 4th, 1868); ten to thirty drops of the tincture are to be given at once and repeated at short intervals. Emetics and the stomach-pump are to be used as early as possible. The latter will require the assistance of an anæsthetic as the attempt to insert the tube may excite spasms of the muscles of the throat or general tetanic convulsions.

To allay the irritability of the spinal cord and counteract the spasms, innumerable drugs have been employed, but at present all are superseded by chloral hydrate and chloroform. Liebreich pointed out the value of chloral in allaying the irritation of the spinal centres, and its subsequent use has proved it to be of decided benefit. Thirty grains by the mouth or rectum, or subcutaneously, will often arrest the muscular spasms for half an hour, and then doses of fifteen or twenty grains, to be repeated when indicated, will usually be sufficient to maintain its effect.

The continued use of chloral must be attended with some caution on account of its depressant action. Bromide of potassium is often combined with chloral, and some cases have been treated by it alone. Its action upon the spinal centres is similar, but it is slower and in every way inferior. Four drachms should be given at once and repeated in doses of one drachm. Chloroform or ether, with or without chloral, is indispensable. Its effect is rapid and its administration may be regulated in accordance with the severity of the spasms. If necessary the patient may be kept under its influence for hours. Chloroform has been given internally with apparent success (*Philadelphia Medical Times*, February 13th, 1873). One drachm was given every hour to a boy, fifteen years of age, who had taken two grains of strychnine. Opium and its alkaloids have been employed with success in many instances. It reduces reflex action but is slow in action and its effects are weak compared with those of the poison. Belladonna, Calabar bean, cannabis indica, and curare have been recommended on account of their antagonistic effect upon the motor centres, but they have not proved serviceable enough to depend upon. Tobacco has been of use in relaxing the muscular spasms and in an emergency an infusion may prove of much value. Artificial respiration may be resorted to if asphyxia is threatened or if the respiration is impeded during the intervals. Inhalations of oxygen will be indicated in the same conditions; their beneficial effect is manifested not alone in the improved state of the blood, but the oxygen is also supposed to act directly upon the poison and mitigate its action. In addition to the special treatment the condition of the patient and his surroundings must be favorable. Perfect rest and quiet must be secured, unnecessary attendants and excitable friends excluded, and all movements must be slow and deliberate. It has been found that continuous movements and sounds may be present without influencing the patient, whereas the slightest sudden starting will bring on the convulsive attacks.

The *post-mortem examination* furnishes very little information as to the cause of death. Rigor mortis begins early and may persist for a prolonged period. It has frequently been noticed several days after death, and Haines, in Hamilton's "System of Legal Medicine," mentions a case in which it was present five months after death. The rigidity, however, is not always present, and its absence does not negative strychnine poisoning. The examination of the stomach may reveal some traces of the poison, if the powder or extract has been taken. The other organs are congested as in death from asphyxia. A congestion of the cord may be present but is very uncertain. A chemical examination of the tissues is the only certain method of determining the presence of the poison. The alkaloid is very persistent and remains for many years without decomposition.

It has been detected in preserved specimens after several years, and numerous instances are recorded in which it has been detected in bodies exhumed many months after burial. The response of certain ptomaines to the tests for strychnine has been pointed out by Vaughan. Failure to detect the poison is not infrequent even in the hands of the most expert chemists, the minuteness of the quantity and its rapid excretion causing it to escape detection. The poison may be present in all the tissues of the body, and in the urine, bile, saliva, and other secretions. The liver and spinal cord are generally selected as the most favorable sites for its detection. The urine also should always be preserved for examination.

Veratrum.

The genus Veratrum, order Liliaceæ, closely allied to colchicum, contains numerous species widely scattered over the globe. *Veratrum album*, Linn., common throughout Europe and Asia is almost identical with *Veratrum viride*, Ait., which is the form found in North America. *Asagræa officinalis*, Lind., also named *Veratrum officinalis*, Schl., is the species common in Mexico and South America.

Veratrum viride is found throughout the temperate parts of North America, extending from the Southern States to Alaska. It grows along streams and in damp places, the plant springing annually from a perennial rhizome. The stem is simple, erect, two to four feet high, bearing numerous three-ranked leaves, and terminating in a dense spreading panicle of yellowish-green flowers. The leaves are broadly oval pointed, sessile, the bases clasping the stem. The flowers are monœciously polygamus, the perianth is formed of six narrow pointed sepals, moderately spreading, entirely free from the ovary. Fruit ripens as a three-celled follicle containing many seeds.

The rhizome is official in the United States and British Pharmacopœias; the latter also recognizes the seeds of Asagræa officinalis under the title of sabadilla. The latter is official only for the production of veratrine. The United States Pharmacopœial preparations are a fluid extract, 1 in 1 part, and a tincture, 1 in 2; the British Pharmacopœial tincture is 1 in 4. Veratrine is official in both, its preparations in the United States Pharmacopœia being an oleate, two per cent., and ointment, four per cent. of the alkaloid. The British Pharmacopœial ointment is much weaker, one grain to the drachm. The white hellebore rhizome is the official drug in Germany and France.

The active principles of the several plants were formerly looked upon as being similar; they are now known to consist of several alkaloids which have been differently named by the various chemists. *Veratrine* is the chief constituent of sabadilla and exists only in small quantities in veratrum, *jervine* being the principal constituent of the latter. The physiological action of both, however, is similar, and the veratrine of commerce generally consists of the two alkaloids. With them are associated *veratroidine*, *cevadine*, and *cevadilline*, which exist in very small amounts and are of no practical importance.

Veratrine is a dull white powder, without smell, having a bitter, acrid taste and leaves a sense of tingling and numbness on the tongue. Alcohol, ether, and chloroform dissolve it readily, but it is only slightly soluble in water.

Veratrine is a powerful motor depressant and muscle poison. It also exercises a local irritant action which may be very severe. It has no action upon the brain nor upon the sensory nerves. The local irritation is evident when it is brought in contact with the skin, causing redness and irritation. Upon the mucous membrane of the mouth, stomach, and intestinal tract it may be so severe as to produce inflammation and ulceration of the parts. There are at first heat and burning of the mouth, throat, and stomach, sneezing, salivation, nausea, vomiting, pain, and diarrhœa, and ultimately all the symptoms of gastro-enteritis. After absorption its action as a direct muscle poison, in addition to the depression of the motor centres, causes great muscular weakness and depression. The muscular contraction is slow and prolonged and uncontrollable, simulating a mild tetanic spasm, then gradually becomes weaker and finally ceases. This is equally directed to the voluntary and involuntary muscles, the heart and blood-vessels becoming paralyzed as well as the body and limbs. The pulse becomes slow and feeble and the respirations grow more and more shallow, death taking place from cardiac syncope. Convulsions and coma sometimes precede death, arising in a measure from the accumulation of carbonic acid in the blood. In some instances the fatal termination is due to peritonitis following the local irritant action.

The poison is rapidly absorbed and the symptoms begin quickly, but the course is prolonged and may continue for some days. When the poisoning is severe, death may take place in a few hours. Fatal cases are not common. Blyth refers to a case in which an infant died from the application of powdered sabadilla to the head for the purpose of destroying pediculi, and to two cases of chronic poisoning terminating fatally after a lapse of nine and eleven weeks, during which time small doses had been continuously given. Haines states that a fatal result has followed the ingestion of twenty grains of the powdered veratrum in one instance, and of a teaspoonful of the tincture in another. Blyth mentions a death from eighteen grains of the rhizome. Very large quantities have been recovered from, and alarming symptoms have arisen from small doses.

An adult took half an ounce of powdered rhizome which gave rise to burning in the throat, pain in the stomach and bowels, depressing nausea, and purging. Recovery rapidly occurred (*Lancet*, II., 1857, 9).

An adult took one drachm of the tincture, equal to twelve grains of the powder. In a short time the patient fell into a collapse, with pain in the stomach, but no purging. Next morning he had recovered (*Medical Times and Gazette*, 1863, January 3d).

The *treatment* consists in removing from the stomach any poison that may remain, and the administration of demulcent drinks and opium to allay the local irritation. The depression and the enfeebled heart and respiration call for stimulants, alcohol, coffee, camphor. Caffeine, strychnine, and digitalis will also prove valuable for their stimulating action upon the heart and respiration. Rest and quiet in the recumbent position must be maintained and heat should be applied to the extremities and surface.

ACIDS.

Sulphuric Acid.

Sulphuric-acid poisoning is not of frequent occurrence in this country, but in Europe the number of deaths from this cause at times has been remarkable. In England they have formed 5.9 per cent. of the deaths from poison, in France 5.5, in Prussia 10.6, in Vienna 43.3, and in Berlin as high as 50 per cent. (Falc). More fatalities are caused by this acid than by any other, and it is more often the cause of accidents than the others, the absence of any fumes or other distinctive characters probably accounting for this frequency. The greater number of cases of poisoning are accidental, the acid having been generally taken in mistake for other fluids or having been left within the reach of young children. Instances have occurred in which it has been added to enemata and to vaginal washes with fatal results (Blyth). A number of cases are recorded in which it has been taken with suicidal intent, but its criminal administration is rare. The victims are generally children, but in some instances adults have been deliberately poisoned and in one case, to avert suspicion, the poison was poured into the ear.

Sulphuric acid is a powerful corrosive and its action as a toxic agent is entirely local, any effect upon the system being the result of the local destruction of tissue. The strong acid is very hygroscopic, increasing its bulk one-fourth by the absorption of moisture. When brought in contact with living tissues this same affinity for water is exhibited and it immediately absorbs the fluid contained in the part, and alters or destroys all fibrous or muscular tissue.

No tissue can withstand this corrosive action, and any brought in contact with the acid may become entirely destroyed and rendered unrecognizable. It has been found that solutions of the acid with a specific gravity above 1.574 dissolve the muscular tissue, changing it into a jelly-like mass which becomes of a blackish-red color. With a specific gravity below 1.574 the solution dissolves only the areolar tissue that is mingled with the muscle, causing the whole to change into a thick pulp of a whitish hue. Any solution above 1.250 precipitates albumin, and when in excess dissolves it (Ziemssen). The specific gravity of the official acid is 1.835 and all acids of commerce approach this strength. The dilute acid and the aromatic acid of the pharmacopœia are weak solutions with little or no toxic action. The specific gravity of the former is about 1.094 and the latter 0.911. When the acid has been swallowed, the condition varies from a whitening and thickening of the mucous membrane to extensive destruction of the tissues. The tongue may be swollen and more or less disorganized and necrosed. The effects of the acid may continue throughout the pharynx and œsophagus into the stomach and in many instances the destructive action has extended into the duodenum. The epiglottis and larynx may be implicated. The affected parts are soft and friable, and in twenty-four or thirty-six hours separate more or less completely as a necrosed mass, frequently being expelled as casts of the part. The under surface remains red and ecchymosed and repairs by granulation if the destruction has been deep. The walls of the stomach are blackened and friable and perforation is frequent. The acid may also penetrate to neighboring vessels, coagulating and blackening the blood. The depth of the destructive process depends upon the concentration of the acid.

The *symptoms* are intense pain in the mouth, throat, and stomach, accompanied by severe reflex retching, vomiting, and spasmodic respiratory efforts, profuse salivation, with extreme distress and prostration. The vomited matters consist of the contents of the stomach, mucus, and particles of necrosed tissue, all being strongly acid and corroded. Should the larynx be affected, the swelling of the part causes difficulty in breathing which may be so severe as to threaten asphyxia and death. Accompanying the local symptoms the system suffers from shock: the pulse becomes weak, the surface and extremities are cold and clammy, and, if the condition is extreme, convulsions, coma, and death may follow rapidly.

The course varies with the extent and location of the injury. In mild cases the local pain and distress continue for some days, the dead tissue is gradually thrown off, and recovery follows, but in other cases death may take place rapidly from the extensiveness of the destructive action and shock, or from occlusion of the larynx. In the greater number of fatal cases it has been found that death takes place in about twenty-four or thirty-six hours, at a time when the tissues are sloughing and the exhaustion is extreme. During this period there may have been symptoms of perforation and peritonitis, and other inflammatory manifestations. If the patient survives this period the system generally recovers from the immediate effects of the poison, repair of the parts commences, and the ultimate termination depends upon the tissue affected and the extent of the contraction or stricture that follows. Occlusion of the œsophagus frequently causes death long after apparent recovery, and strictures and deformities of the stomach and throat are common.

Very small quantities may be sufficient to produce death. Twenty drops have proved fatal to a child (Taylor), and sixty to an adult (Christison). Blyth relates a case in which two mouthfuls of a solution of one part of acid to four of water proved fatal to an adult. A few drops of the acid brought in contact with the larynx would prove very serious, while on the other hand very severe destruction of the tissues of the mouth may cause alarming but not dangerous symptoms.

The *treatment* consists in neutralizing the acid with alkalies and diluting it with copious draughts of water. Magnesia, lime, and carbonate of soda are the most serviceable. The stomach-pump and emetics are to be avoided. Milk and mucilaginous drinks and antiseptic solutions are to be administered freely after the first effects are counteracted.

Nitric Acid.

Nitric acid is second only to sulphuric acid in the severity of its action, and, as with the stronger acid, the tissues are deeply and rapidly destroyed, the effect extending to the stomach and duodenum. Perforation may also take place. Blyth gives a valuable description of numerous specimens in the various London museums illustrating the results of nitric-acid poisoning. The skin is also destroyed and colored a characteristic yellow tinge; the same discoloration is present in all the parts attacked and serves as a guide to distinguish the action of nitric from that of other acids. Bromine and iodine produce a stain which might be mistaken for that caused by nitric acid, but both are easily removed by a little caustic potash, while nitric-acid stains become a brighter orange by the action of the alkali.

The *symptoms* are those associated with the local destruction of tissue and are similar to those of sulphuric-acid poisoning. In addition there is the production of an abundant supply of gas, which causes distressing distention of the stomach and intestines and painful eructations. The course is rapid, the result depending upon the strength and amount of the acid. Deaths have taken place in a few minutes (Taylor) and many instances are recorded of a fatal termination within a few hours. The period of twenty-four or thirty hours is, as in poisoning by sulphuric acid, a critical period, and many deaths also occur from the remote effects. Two drachms have caused death (Taylor) and half an ounce has repeatedly been followed by a fatal result. As with other corrosives a small quantity to the larynx may prove fatal, while a very much larger quantity may produce severe effects in less important parts without serious results.

The *treatment* is the same as that for sulphuric-acid poisoning—alkalies in solution to neutralize, and fluids to dilute the acid, soothing and bland drinks, and frequent thorough irrigation of the mouth and affected parts.

Numerous deaths have followed the inhalation of the fumes of nitric acid, which act as an irritant to the bronchial mucous membranes. There may be an immediate spasm of the glottis and fatal asphyxia, or a broncho-pneumonia and œdema of the lungs may follow. Blyth refers to a report (*Chemical News*, 1863, 132) of the case of two young men who broke a jar of nitric acid and inhaled the fumes for some time; one died in ten hours, the other in twenty-four. Pellew, in Hamilton's "System of Legal Medicine," also refers to the case reported in the *Boston Medical and Surgical Journal*, 1884, p. 496, of four men who inhaled the fumes in a similar way. One had an attack of pneumonia in a few days, another in three hours felt a constriction of the chest with painful inspiration; he remained ill for three or four days with painful respiration and prostration. A third had catarrh of the upper air-passages for three weeks. The fourth was longer exposed than the others and after suffering from painful irritation of the pulmonary organs with great prostration died in about thirty hours from œdema of the lungs.

Hydrochloric Acid.

Hydrochloric acid is a much weaker acid than sulphuric or nitric and its effects when swallowed are less severe. Instead of the extensive and deep destruction of tissue, the parts become whitish, or whitish-brown; in some instances they are disorganized, but not to any great depth. The acid converts the superficial tissues into a swollen, structureless, slimy mass rather than causes a corroded slough. It does not exercise any corrosive action upon the skin. The walls of the stomach are not eroded and perforation rarely occurs.

The *symptoms* are similar to those following the ingestion of other corrosives but not so intense, and the destructive effects not being so severe, the tendency to recovery is more favorable. In addition to the local irritant action, the noxious fumes produce an irritation of the larynx and lungs and give rise to spasmodic coughing and choking. Numerous deaths have resulted from this cause, in some instances after the ingestion of very small quantities. One drachm has proved fatal in sixteen hours (Ziemssen), and half an ounce in eighteen hours (Taylor). The fatal termination may take place at any time as in the case of other acids, and the remote effects may also be the cause of death.

The *treatment* is the same as in the case of the other acids.

A man, 54 years old, swallowed one ounce of hydrochloric acid. In three hours he was collapsed. Skin was cold and clammy, countenance anxious; pulse small and weak; there was frequent vomiting of dark, viscid matter containing blood. The buccal mucous membrane was whitish with here and there a red patch. He complained of burning in the mouth, throat, and stomach; there was no diarrhœa nor tenesmus. Solution of potash, freely diluted, and lime-water and milk were given freely, but the man continued prostrated, vomited continually, and died in seventeen hours. Post-mortem: the mucous membrane in the upper parts was highly congested, in the lower half of the œsophagus it was black, thickened, and contracted. Just before the passage through the diaphragm there was a perforation in the posterior wall the size of a shilling; the tissues around were black and pulpy. The mucous membrane of the stomach and of about five inches of the duodenum was similar to that in the lower part of the œsophagus. There was a perforation in the stomach and one in the duodenum (*Lancet,* 1884, I., 65).

Adult, male; accidentally swallowed one ounce of pure hydrochloric acid. There was immediately pain in the mouth and throat, reaching down the gullet to the stomach; retching and vomiting followed, and continued severely, being marked with blood. The tongue, soft palate, fauces, and back of the throat became yellowish; there were no marks on the chin or cheeks. Temperature 96°; the man was given a solution of carbonate of soda, one drachm to four ounces, together with white of egg and nutrient enemata. On the tenth day he vomited a quantity of blood and offensive matter and improved more rapidly afterward. Feeding by the mouth was commenced on the eighteenth day, and the man left hospital feeling quite well in about one month after the accident. One month later he returned with evidence of obstruction of the pylorus and died in a few days.

Post-mortem examination showed general contraction of the stomach, the mucous membrane near the pylorus presented cicatrices, the pyloric wall and surrounding tissues were thickened and altered; a small probe only could be passed through the lumen of the pylorus (*Lancet*, 1890, II., 797).

Carbolic Acid.

Carbolic acid, or phenol, C_6H_5HO, is one of the numerous constituents of coal-tar, from which it is separated by fractional distillation. It is obtained at temperatures between 329° F. and 374° F. Associated with carbolic acid in the distillate is a varying amount of cresylic acid or cresol, and much of the impure carbolic acid of commerce is made up of these two compounds; some specimens being almost entirely cresol. As cresol is devoid of the toxic action of carbolic acid, the impure acid is much less poisonous, and in many instances large doses have been taken without any serious result.

Carbolic acid when pure is a crystalline solid, forming in white or colorless needles, with its peculiar odor and a hot burning taste. On exposure it may acquire a pinkish tinge. It rapidly absorbs moisture and becomes a deliquescent mass, and with sufficient moisture a liquid. It melts at 106° F.; does not redden litmus paper. Cresol differs from it in not crystallizing at ordinary temperatures; its boiling point is higher, about 400° F., and it is less soluble in water, ammonia, glycerin, or solution of soda. Creasote, which is a mixture of cresol and guaiacol, and is almost without any poisonous action, is of a lower specific gravity, does not crystallize, and is also less soluble in ammonia, and not at all soluble in glycerin. It does not coagulate albumen. The United States Pharmacopœia requires carbolic acid to stand the following tests: "The aqueous solution yields with bromine water a white precipitate, which at first redissolves, but becomes permanent as more of the reagent is added, and appears crystalline when viewed under the microscope. On adding to 10 c.c. of a one-per-cent. solution one drop of ferric chloride test solution the liquid acquires a violet-blue color which is permanent; and on adding carbolic acid to albumen or collodion, coagulation takes place (difference from creasote). One volume of cold liquefied carbolic acid (rendered liquid by the addition of eight per cent. of water) forms, with one volume of glycerin, a clear liquid which is not rendered turbid by the addition of three volumes of water (absence of cresol and creasote)."

When applied to the skin, carbolic acid at once combines with the albuminous tissues and forms a white eschar, which may penetrate to some depth and destroy the parts, a zone of inflammatory action marking the limits of the dead tissue. Upon mucous membranes its effects are more severe, and in cases in which the pure acid is swallowed the local action may be sufficient to cause death. When recovery takes place the after-effects are not severe and strictures rarely follow.

Carbolic acid is absorbed from the surface of the body, from wounds, by the pulmonary tissue when inhaled, and from all the mucous membranes. Its course after absorption is unknown. It may be detected in the blood for a time unchanged, but it is rapidly decomposed and the products of the change are partially excreted with the urine, saliva, perspiration, and other secretions. It causes a dark, smoky discoloration of the urine which contains the acid as sulphocarbolic acid, the sulphates disappearing some time before the discoloration becomes noticeable or symptoms of poisoning appear. Some observers have detected the odor of carbolic acid in the urine and consider that portions of it are excreted unchanged. By some it is also thought to be excreted from the lungs.

Symptoms.—After absorption the toxic effects are manifested on the nervous system. At first there is a general stimulating action, the pulse becomes stronger, the blood pressure rises, the cerebral and spinal centres are exalted, with convulsions and delirium, and the temperature increases to a marked degree. This state of stimulation is followed by depression and paralysis of the entire nervous system, and death takes place from respiratory and cardiac failure.

The effects are best exemplified in the severe cases when death takes place rapidly. Almost immediately there is nausea and faintness, pallor of the skin and cold sweat, dizziness, and dulness of the senses which deepens into insensibility and coma, with muscular weakness and convulsions ending in prostration. The pulse becomes weak, the respirations shallow and hurried, and death ensues, sometimes suddenly from syncope, at other times more gradually from asphyxia. When the course is less rapid the sequence of symptoms is more prolonged and may be preceded by the primary stimulating effects. In addition to the constitutional disturbance there are also the local effects and gastro-intestinal irritation, which may be severe; or the acid may have implicated the larynx and produced an additional train of symptoms. In very mild cases, in which the absorption of the poison is slow and prolonged, as in the case of wounds and extensive surfaces dressed with solutions of carbolic acid, there may be simply restlessness, headache, increased temperature, debility, and diminished muscular power—symptoms which may very readily be mistaken for diseased states of the system.

The course may be slow and prolonged for days when the absorption is gradual, and the symptoms may persist after the cause is removed, but in cases in which the poison is swallowed it is much more rapid. In many instances the patient has fallen unconscious within the space of one minute and died in a few minutes. More frequently death does not take place until from one-half to two hours have elapsed, or life may be prolonged for several hours or days.

The *fatal dose* is uncertain, but half an ounce is considered a dangerous quantity and many cases are recorded in which this has caused death. As small an amount as one drachm has also proved fatal (Taylor). Very severe symptoms have followed the use of carbolic-acid dressings, and also of carbolic solutions in irrigating abscess cavities and wounds. The employment of a strong solution as a lotion has also, in several instances, proved fatal (Haines, in Hamilton's "System of Legal Medicine").

Post-mortem examination does not furnish any clew to the cause of death except that it may bring to light evidence of the effects of the poison upon the mucous membrane of the mouth and stomach; the presence of the acid in the stomach or bowels may also be detected by means of the odor. There may be patches of brownish or white wrinkled skin on the face and hands, the lining membrane of the digestive tract may be whitened, thickened, and shrivelled, with localized areas of destroyed tissue. When death has been gradual the organs present the appearances resulting from asphyxia, but when it has been sudden no change is to be noted. The urine may furnish some evidence. The presence of free acid may be detected if it remains in the stomach, but, after absorption, tests have to be directed to the products of its decomposition, and their presence may be indicated with a fair amount of accuracy.

An infant, seven weeks old, was ill with varicella with one pustule, which was surrounded by an erysipelatous area. A two-per-cent. solution of carbolic acid was kept applied by means of a compress. The following morning symptoms of poisoning were present. The temperature rose, the urine was dark; there were sweats, vomiting, contracted pupils, spasmodic twitching of the eyelids, and slow respiration. Death occurred in twenty-three hours from the first application (Blyth).

A boy, aged 5, was given an enema of about 18 ounces of 1 to 60 solution of carbolic acid, equal to 144 grains. In a few minutes he became unconscious without premonitory symptoms. He remained insensible and in a state of collapse for several hours when he died (*Lancet*, May 19th, 1883).

The *treatment* consists in removing the source of the poison if it is external, or, if it has been taken internally, in washing out the stomach carefully with the aid of the pump. Emesis will be difficult to procure, and is not so effective. After removing all traces of the poison, milk, oil, and demulcent drinks may be administered; but the use of oil is prohibited while any poison is in the stomach, as it is supposed to promote its absorption. The most useful antidote is suggested by the affinity of carbolic acid for sulphates and the formation of the harmless sulpho-carbolic acid. Sulphate of magnesia, sulphate of soda in solution, or sulphuric acid diluted, may be administered freely, and in many instances this has been found of benefit. The same reaction is supposed to take place in the blood when the sulphates are absorbed. Saccharated syrup of lime has also been proposed to neutralize the acid. The lime readily combines with carbolic acid to form a soluble and harmless salt. Lime-water is not of much service as the amount of lime is too small to be of any value. Atropine is also used with the object of stimulating the respiration and heart. Opium may prove of use to relieve the pain.

Hydrocyanic Acid.

Hydrocyanic acid, HCN, is one of the most powerful poisons, and one of the most common causes of death from poisoning. The pure acid is rarely met with. It is a colorless, transparent, volatile liquid, with a specific gravity 0.6969, possessing the characteristic taste and odor of bitter almonds. It is soluble in water and alcohol. The vapor is exceedingly poisonous and rapidly causes death. The dilute hydrocyanic acid of the pharmacopœia is the form in which it is kept. This contains two per cent. of the anhydrous acid. Formerly there were solutions of various strength, but at present the only other one is Scheele's, which contains five per cent. of the acid. Dilute hydrocyanic acid is also transparent, colorless, and volatile, specific gravity 0.997. Owing to the volatile nature of the acid, and to the fact that it is readily decomposed when exposed to the light, very many specimens have greatly deteriorated and lost much of their virtue. The following are the official tests to determine their strength: "To ascertain the percentage strength, mix in a flask (of the capacity of 100 c.c.) 0.27 gm. of the acid with sufficient water and magnesia to make an opaque mixture of about 10 c.c. Add to this two or three drops of potassium chromate test solution, and then, from a burette, silver nitrate decinormal volumetric solution, until a red tint is produced which does not again disappear on shaking. Each cubic centrimetre of silver nitrate volumetric solution used indicates one per cent. of absolute hydrocyanic acid" (United States Pharmacopœia). The British Pharmacopœia test states that 270 grains of a two-per-cent. solution, made alkaline by solution of soda, will require 1,000 grain measures of the volumetric solution of silver nitrate to be added before a permanent deposit begins to form.

Potassium cyanide, KCN, contains at least forty per cent. of the anhydrous acid, and is as frequent a source of accidents as the dilute acid. It occurs in white, opaque, amorphous, deliquescent masses; without odor in dry air, but with moisture exhaling the characteristic fumes; soluble in two parts of water. It rapidly decomposes on exposure and many samples have been found to contain a large percentage of impurities.

Oil of bitter almonds is a peculiar vegetable product containing ten to fifteen per cent. of hydrocyanic acid, which is formed in the kernel of the nut by the action of *emulsin* upon *amygdalin*. It is volatile and obtained by distillation as "a clear, colorless or yellowish, thin, and strongly refractive liquid, having a peculiar aromatic odor and a bitter and burning taste. Specific gravity 1.060 to 1.070." It is a mixture of hydrocyanic acid and benzoic aldehyde, C_6H_5COH, and has all the poisonous properties of the acid. The acid may be removed and the oil freely used for flavoring purposes without fear of any poisonous action.

An artificial oil of bitter almonds is also prepared, which is *nitrobenzole*, $C_6H_5NO_2$, having a poisonous action equal to that of the crude oil of bitter almonds. It is an oily, yellowish liquid, with the odor of bitter almonds, but has a very sweet taste. Specific gravity, 1.186. During its preparation headache and dizziness are produced and many instances of death have followed its use. In cases of poisoning there is headache, muscular depression, consciousness becomes dulled, the pulse and respiration become weak and irregular, the lips, extremities, and surface of the body become bluish; the pupils are dilated; paralysis and coma follow, and death occurs from asphyxia. After death the blood is of a dark chocolate-brown color. Fifteen drops have proved fatal (Taylor). The symptoms of poisoning are very similar to those of aniline, to which the substance is closely allied.

Hydrocyanic acid is a powerful depressant and causes death by its combined action upon the blood and upon the centres presiding over the heart and respiration. The course of the poison after absorption is unknown as it becomes decomposed and cannot be detected in the excretions. It immediately promotes changes in the hæmoglobin of the blood, hindering its power of absorbing oxygen. At first the blood in the veins and in the right side of the heart becomes bright red and spectroscopic analysis shows the bands of oxyhæmoglobin. This effect, however, is very transient, and all the blood becomes dark and venous in character and exhibits with the spectroscope only the bands of deoxidized hæmoglobin. After death the blood is fluid and dark or bluish-black in color. It is now generally accepted that the poison exercises a direct poisonous action upon the nervous system, paralyzing both the terminal fibres and the centres. This is especially directed to the cardiac and respiratory centres. In the majority of cases the respiration is chiefly affected and death is due to asphyxia, but in many cases death is apparently due to cardiac syncope. Experimental work has shown that the poison applied to the heart immediately paralyzes its action and all power to respond to stimuli is lost. The whole question of the manner in which the effects of this poison are produced is still unsolved and has yet to be explained.

The *symptoms* of poisoning vary with the quantity taken. When the quantity is not great there arise a sense of uneasiness, headache, difficulty in respiration, and constriction of the chest-walls, sometimes salivation with nausea and vomiting. The headache increases, with a sense of fulness in the head, giddiness, confusion, vertigo, numbness of the extremities, and failure of locomotion. Respirations become slow and labored, the expiratory movement being marked. The face becomes pale, expression anxious, eyes prominent, and the jaws set. Convulsions of a tetanic character may appear; unconsciousness and paralysis follow, a cyanotic state of the blood is evident, and death results from asphyxia. When the dose is excessive the patient may suddenly be prostrated, unconscious, and convulsed, and death may take place in a few seconds; or the unconsciousness and paralysis may persist without convulsions, the jaws being rigid, with much frothy mucus about the lips; the breathing is slow, pulse almost imperceptible, skin pallid or cyanotic, eyes staring; the length of time until the muscles relax and the respiration fails entirely varies. When the poison is inhaled it acts with equal rapidity, as the lungs readily absorb the volatile fumes.

The course of hydrocyanic-acid poisoning is very rapid, death usually taking place within a few minutes, rarely later than an hour. So sudden is it in many cases that it is described as having killed instantaneously, but there is always an appreciable interval which Blyth estimates to be at least ten seconds. This fact is of medicolegal interest, as it often becomes important to determine whether certain acts may have been performed before death. On the other hand there are many instances where very large doses have been taken, in which sufficient time has elapsed before death to allow the patient to walk some distance and perform many voluntary acts. Such cases are, however, rare, as, even if the poison fails to prove fatal, the system is so rapidly influenced that voluntary movements are annulled. When the tendency is to recovery the improvement is first noted in the respiratory efforts and in the color of the skin, and the unfavorable symptoms rapidly pass away.

It is very generally accepted by all authorities that one grain of the anhydrous acid, or its equivalent, is the minimum fatal dose. This would equal 2.4 grains of the cyanide of potassium, or 50 minims of the dilute acid, or twenty to thirty drops of the oil of bitter almonds.

Adult male, aged 22 years, swallowed one-half ounce of dilute hydrocyanic acid, and was found unconscious on a bench, with eyes upturned and limbs relaxed. When moved he had a slight clonic convulsion; the pupils were dilated, pulse 86, respirations shallow and sighing, 40 to 50; the breath had the odor of almonds. The pulse ran up to 112, the dyspnœa increased, and trismus was marked. Lavage was performed, stimulants subcutaneously injected and faradization practised. In twenty minutes there was improvement and in one and one-half hours the man was out of danger, but remained unconscious for two hours. The acid was detected in the urine, which also contained albumin and sugar (*American Journal of the Medical Sciences*, July, 1890).

A woman, 31 years of age, ate a quantity of bitter almonds with suicidal intent. In a few minutes she complained of sickness and faintness, and vomited. In ten minutes she fell senseless in a convulsion. An hour after she was insensible, with eyes rolled upward, hands clenched, breathing rattling, pulse very slow. Died in one and one-half hours after eating the poison. Post-mortem: Organs healthy, all except the liver; exhaled the odor of bitter almonds. The right side of the heart was full of dark blood; the left side was empty. Both lungs were rich in blood. The stomach, which was not inflamed, contained yellow fluid. At the most dependent part the mucous membrane was swollen and denuded of epithelium; the mucous membrane of the duodenum was also swollen and slightly red. The residue of the almonds was present. The amount taken was calculated to have been about 1,200 grains, equal to 43 grains of amygdalin, or 2.5 grains of pure acid (Blyth).

Treatment.—In severe cases there is rarely an opportunity to attempt to counteract the effects of the poison, but in milder poisoning, efforts must be made to prevent the asphyxia and allow the system to recover itself. Cold affusions to the head and spine, artificial respiration, and electricity must be employed to maintain the purity of the blood. Hydrogen peroxide is also recommended for the same purpose and inhalations of oxygen should be used. Atropine has been suggested upon theoretical grounds by those who think the respiratory failure is due to the irritation of the peripheral branches of the vagus, as the atropine shows an antagonistic action by paralyzing the same terminations. The alkaloid, however, has not proved of much benefit, and the theory upon which its use is based has not been accepted. Attempts should also be made to wash out the stomach and remove any trace of the poison that might remain unabsorbed. Chlorine and ammonia are recommended on account of their power of dilating the capillaries and stimulating the respiratory and circulatory organs. The ammonia may be inhaled or injected into a vein. Chlorine water or solution of chlorinated lime or soda should be given freely. The only chemical antidote given to neutralize the acid in the stomach is a salt of iron which combines with the acid to form insoluble prussian blue. This has been suggested by Messrs. T. and H. Smith, of Edinburgh. The following is the formula they recommend: "From one to two drachms of magnesia made into a cream with water are to be administered, and afterward 16 minims of solution of perchloride of iron and 12½ grains of ferrous sulphate in solution in water. These quantities will neutralize 100 grains of the dilute acid" (United States Dispensatory, 1890).

The *post-mortem* examination furnishes very little information unless the characteristic odor is present. In some cases the acid has caused a redness of the mucous lining of the stomach; if the cyanide of potassium is the cause this will be very well marked. The blood also may furnish some evidence, but as a rule the condition of the blood and the organs differs very little from their appearance in asphyxia from any cause. The volatile character of the acid makes it improbable that any trace of the poison will be obtained after death, yet it has been detected very frequently after many days, and in some cases after burial for months.

Oxalic Acid.

Oxalic acid is a very active poison and is answerable for a great number of deaths. The reports of the Registrar-General of Great Britain show it to be one of the most frequent of causes of death from poison, and in this country such cases frequently occur. The greater number are attempts at suicide, and the remainder are the result of accidents. Of the latter a great many arise from the taking of "*salts of lemon*," which in most cases is composed almost entirely of oxalic acid.

Its action is directed to the cerebral and spinal nervous system, upon which it acts as a depressant, and causes death by a failure of the heart and circulation. Its physiological action is not thoroughly understood, and an uncertainty exists as to its mode of producing its fatal effects. By some it is thought to exert a direct poisonous action upon the heart, but the more generally accepted view is that it paralyzes the nerve centres and gives rise to complete exhaustion. In addition to its constitutional effects it has a local irritant action which in concentrated solutions may become corrosive, simulating the milder effects of the mineral acids or alkalies. The mucous membrane may be simply reddened or of whitish or yellowish color, thickened and denuded of its epithelium.

The *symptoms* of poisoning vary considerably. The local distress may be severe, with pain extending from the mouth to the stomach, constriction of the throat, dyspnœa, nausea, retching, and vomiting of bloody matter, great tenderness, and cramps; or, when the solution is well diluted, there may not be any symptoms referable to its local action.

Its absorption is rapid and its effects are almost immediately detected. The countenance becomes pale and anxious and the surface is covered with a cold, clammy perspiration. The pulse becomes frequent, feeble, and intermittent, the respirations shallow and labored; spasms and convulsions may occur, also stupor and unconsciousness. In fatal cases a condition of cyanosis follows upon the collapse and hastens death. When the dose has been excessive the patient may suddenly become insensible and die in a few minutes.

The course is rapid and a fatal termination may take place in ten minutes (*Chemical News*, April 24th, 1868). The majority of deaths take place within half an hour or one hour, in other cases the symptoms persist for ten or twelve hours. The acid is rapidly excreted and the commencement of favorable symptoms is seen early. The patient rapidly passes from danger, but convalescence may be very protracted as the injury to the nervous system, as well as the local irritation, may give rise to distressing symptoms which persist for some time. Death has taken place as late as the second week from inanition, the result of the destruction of the mucous membrane of the stomach (*Edinburgh Medical Journal*, 1818, 607).

As with all poisons the quantity that is likely to prove fatal is somewhat uncertain. Although very much larger doses have been recovered from, one-half ounce is usually fatal, unless the vomiting removes a portion of it before absorption; one drachm is the smallest dose that has proved fatal (Taylor).

A man, 45 years old, took about one ounce of oxalic acid with suicidal intent. Vomiting had taken place when he was seen one-half hour later. There was great distress, constant vomiting, pain in the throat and stomach, and great difficulty in swallowing owing to the pain and swelling of the parts. Some chalk and milk were given, but the vomiting continued. The man became semi-unconscious and when roused was excited and delirious. The pulse was very weak, and enemata of brandy were given. For a time he complained of severe pain in the hand, then in the eyes, head, and feet. There were also epileptiform convulsions, four during the first hour. In four or five hours the man was easier and improving, but restlessness, vomiting, weakness, and local pain continued for eight or ten days. When last seen, twelve days after the poisoning, there was still tenderness (*Montreal Medical Journal,* August, 1890).

The *treatment* consists in evacuating the stomach, if vomiting has not taken place. Water should not be given unless vomiting is taking place, as it only dissolves the acid and hastens its absorption. The one antidote is solution of lime which combines with the acid to form an insoluble oxalate. The saccharated solution of lime is to be preferred, or a simple mixture of lime and water. Magnesia also forms an insoluble oxalate, but the carbonates of sodium and potassium are not to be used, as they form soluble oxalates, which are almost as poisonous as the pure acid. Ringer has advocated the employment of the chloride of lime, in order to have it absorbed and act upon the poison that it may meet with in the blood. It has been customary to advise the immediate use of scrapings from wall plaster as a ready source of the antidote. After the stomach has been cleaned, water and diuretics may be useful in promoting the elimination of the poison. Opium for the pain, and stimulants should be given, as indicated.

The *post-mortem* appearances do not furnish any evidence, unless a concentrated solution of the acid has been taken. If the strength is less than one part in twenty there is little if any corrosive action. In some cases the mucous membrane may be greatly congested and the epithelium softened and pulpy, but the effects are not characteristic and may easily be mistaken for post-mortem maceration. The acid or its salts may be detected after death in the contents of the stomach and intestines, blood, liver, kidneys, and urine. The microscope will also detect its presence by the characteristic oxalates.

ALKALIES.

Potassa and Soda.

Potassa and soda have powerful escharotic properties and greatly resemble the corrosive acids in the effects they produce when brought in contact with the tissues. Soda is somewhat weaker in its action than potassa, but both have a great affinity for water and are very deliquescent. The potash salt forms a much more fluid compound and in consequence its cauterant action penetrates deeper and more extensively. When swallowed they attack all the tissues, combining with the albuminates and fats to form definite compounds, which dissolve in an abundance of fluid. The parts become whitened and swollen, and then subsequently soften and are expelled as disorganized masses. All tissues may be destroyed. The stomach becomes friable and perforation may occur; the caustic action extends into the intestines. Upon the skin the same corrosive effects are produced.

The *symptoms* are such as would follow the local irritation, pain extending into the stomach, retching and vomiting, difficult breathing, huskiness of the voice, and choking sensations; the lips and tongue become swollen and discolored. Accompanying the local symptoms there is much prostration with a tendency to collapse, which may be increased by the absorption of the alkali. Later the sloughing tissues are cast off and inflammatory action commences. The course of the symptoms is not so rapid as in poisoning by the strong acids or ammonia, three hours being the shortest interval before death (Taylor). Strictures and secondary troubles are not so common as after ingestion of the acids, but they occasionally occur, and many deaths may be traced to this cause. Pellew (Hamilton's "System of Legal Medicine") quotes a case of an adult who took a quantity of caustic potash with suicidal intent. At the end of a week a cast of the œsophagus and larynx was expelled consisting of the mucous, submucous, and portions of the muscular coats; constriction of the passage followed and the patient died in three months.

The *treatment* consists in neutralizing the alkalies with dilute acid drinks, such as vinegar and water, or lemon juice. Oils and fats relieve the pains and distress. Milk, barley-water, and other soothing drinks should also be administered freely. The later stages require to be treated on ordinary surgical principles, antiseptic irrigation and supporting treatment being essential.

Ammonia.

This alkali is much more frequently a cause of poisoning than potassa or soda, and accidents arising from its mistaken use are of common occurrence. Deaths have arisen from inhalations of the gas, from the administation of the liquid, and also from overdoses of the carbonate and muriate. The liquid is the most usual form from which accidents arise, and in its action it combines the effects of the vapor and the fluid. The local irritant action is not so severe as in cases of poisoning by the other alkalies, the escharotic properties are very mild, and there is not the same destruction of tissue. Both the vapor and the liquid, when they reach the mucous membrane, produce a great irritation, with pain, redness, and swelling of the part, which may proceed to vesication. In severe cases the mucous membrane becomes swollen and altered, and a certain amount of sloughing takes place, which may extend from the mouth to the stomach. The vapor being inhaled may produce the same effect upon the laryngeal and bronchial mucous membrane. When the ammonia is freely absorbed, the stimulant action of small doses increases to one of excitation of the brain and spinal cord, which ultimately ends in exhaustion and paralysis of the heart and respiration. In this way ammonia differs from the other alkalies, which have little or no effect beyond that arising from the local action of the poison.

The *symptoms* are intense pain in the mouth, throat, and stomach, with retching and vomiting of mucus and blood. Cough and reflex spasm of the glottis take place at once, the respirations are painful, aphonia is common, and lacrymation and sneezing are always present. The excretion of mucus may be excessive and the lungs become choked. Salivation has been noticed. The pulse and respirations are stronger at first, but ultimately both fail and prostration becomes severe. Convulsions, delirium, and coma may be present. The course is usually toward recovery, the irritation and inflammatory action continuing for a few days, and then gradually subsiding; but fatalities are not uncommon and death may take place early. Christison records a case in which death occurred in four minutes, and Blyth refers to another instance in which death took place within the same time. These early deaths are due to the spasm of the respiratory organ, and the paralyzing action of ammonia upon the cardiac and respiratory centres. As with other corrosive poisons death may take place at any time, from a few minutes to several days, due to the local action and the exhaustion that follows. When the primary effects are recovered from there is not much danger of secondary troubles, unless the corrosive action has been very severe. Deaths from stricture, however, are reported (Taylor). In cases of poisoning by the salts of ammonia, the local irritant action is not so important as the action upon the brain and cord. The latter causes great excitement, with deep inspirations, full pulse, convulsions and delirium, which are quickly replaced by prostration and failure of circulation and respiration. In such cases the local irritant action may be severe but is not fatal.

A man while suffering from delirium tremens drank a mixture of ammonia, olive oil, and turpentine. He was seen an hour later and then had flushed face, congested conjunctivæ; the tongue was swollen and denuded of epithelium and the mouth was swollen; the palate or throat could not be seen. Emetics were given and the man threw off a quantity of blood-stained fluid. In half an hour he began to suffer from dyspnœa which increased in severity and came on in paroxysms. The face became cyanosed; breathing was loud and whistling, and the distress was very great. Tracheotomy was performed and recovery followed slowly (*Lancet*, 1890, II., 1,214).

Four men inhaled ammonia gas for three minutes. One when dragged out was comatose and unconscious and died in fifteen minutes. Another was unconscious, but wildly delirious and unable to stand; he died suddenly in two hours. The third was conscious and walked home; he could swallow and talk, but complained of difficulty in breathing. In about five hours he had an attack of dyspnœa and died suddenly. The fourth suffered from bronchial irritation for some months, but recovered (*St. Louis Medical and Surgical Journal*, 1887, 272).

Adult, aged 54; had been drinking for two weeks. After lunch, by mistake he swallowed a mouthful of liquor ammoniæ. He walked downstairs and then became insensible. Milk was given at once and emetics emptied the stomach; he soon became conscious; the voice was husky but there was no dyspnœa. After three hours the pain was most severe in the epigastrium; vomiting continued every fifteen or twenty minutes, the vomited matter consisting of almost pure blood; the pulse was weak and rapid, features pinched, surface clammy. The following day vomiting and hemorrhage ceased, but there was a continual attempt to clear the throat; stomach would not retain anything. In twenty-four hours he became delirious. This subsided the following day but he became weaker; the irritability of the stomach persisted, and he continued to expectorate very fetid matter. The abdomen was swollen and tympanitic but not tender. The man remained quite sensible until the last. He died of exhaustion ninety-four hours after taking the ammonia (*Canada Medical and Surgical Journal*, X., 449).

The *treatment* consists in the administration of dilute vinegar and lemon juice to neutralize the ammonia. These are to be succeeded by milk, oil, and mucilaginous and bland drinks. Morphine should be given to allay the excitement and delirium and relieve the pain. The after-treatment is to be followed out upon ordinary surgical principles.

CHLORAL HYDRATE.

The employment of chloral is frequently attended with alarming symptoms and many deaths have arisen from its use. In addition to the effect of over-doses, symptoms of poisoning may arise during its therapeutic administration, owing to a personal idiosyncrasy or to some diseased state of the heart or kidneys. The aged are very susceptible to its influence, also those who are debilitated from debauch and excesses, and the most dangerous condition in which it may be administered is that in which the muscular tissue of the heart and vessels is degenerated and fatty.

The action of chloral is chiefly directed to the central nervous system upon which it acts as a paralyzant, it also is supposed to have a direct toxic effect upon the heart itself. It affects first the convolutions and higher centres and gradually descends to the spinal cord, depressing the motor and sensory ganglia and the centres presiding over respiration and circulation. How its action is effected is still uncertain. The early idea of Liebreich, that it was decomposed in the blood and chloroform set free, is not now accepted, the more generally received view being that the drug is carried directly to the tissues and exerts a specific action upon the gray matter of the brain and cord.

The mode of causing death is through respiratory failure, following the paralysis of the nerve centres; but in many cases, where there is a diseased condition of the heart, death is due to cardiac paralysis and frequently occurs as a sudden syncope.

The *symptoms* of an over-dose are a deep and profound sleep which passes into coma, with muscular relaxation, loss of reflex irritability, coldness and paleness of the skin, weak and shallow respirations, and a feeble, intermittent pulse. In many cases death is preceded by a condition of cyanosis.

Chloral is rapidly absorbed, and its effects begin within a few minutes after it has been taken, and death has followed within fifteen minutes (Blyth). Generally the termination is not so rapid and the patient will remain in a comatose state for five or six hours, the length of time greatly depending upon the quantity of poison that has been taken. The fatal dose is very variable. Blyth estimates that two drachms is a very dangerous quantity and that two and a half drachms will almost invariably prove fatal. There are many cases reported in which death has followed much smaller doses. One drachm has frequently caused death, and ten grains have been sufficient in one case (Haines, Hamilton's "System of Legal Medicine"). One ounce is probably the largest dose that has been recovered from (*Chicago Medical Review*, 1882).

There is no post-mortem evidence of any value unless the odor of chloroform is to be detected, and the chance of this is very problematical. The tests applied have this end in view, alkalies being added to the suspected matter that the chloral may be decomposed and chloroform evolved.

The *treatment* consists in removing from the stomach all traces of the poison, by the aid of emetics or the stomach-pump, the exhibition of stimulants, and the application of warmth to the surface and extremities to maintain the body temperature. In addition to these measures the antidotes to be selected are physiological stimulants of the medullary and spinal centres. Strychnine is generally used and many cases are reported in which its action has been most favorable. The hypodermic injection of $\frac{1}{20}$ grain, repeated in fifteen or twenty minutes, will improve the respirations and strengthen the pulse. Picrotoxin in $\frac{1}{20}$ grain doses has recently been highly extolled (*Lancet*, 1891, I., 769). Atropine is one of the antidotes that has been longest employed, its action being to stimulate the respiration. Digitalin has also been recommended. The inhalation of amyl nitrite has proved of service in some instances. The faradic current and artificial respiration should not be neglected if other measures fail to maintain the respiration. Oxygen inhalations should also act beneficially.

ILLUMINATING GAS.

The subject of poisoning by this gas is of the greatest practical importance, owing to the number of accidents and the frequent deaths that may be traced to it. The escape of the gas in sleeping-rooms and among workmen is the most common mode of poisoning; but the gradual contamination of the air of dwellings, by leaking pipes and by defective stoves, is the source of many cases of chronic poisoning, often of a very serious character.

The toxic qualities of the gas arise from the presence of carbon monoxide and certain volatile hydrocarbons. The former, on account of its poisonous character, is of the most importance, the latter being of a secondary consideration in comparison with the more noxious gas. Ordinary illuminating gas contains carbon monoxide in proportions varying from five to nine or ten per cent.; but the more modern gas, known as water-gas, may have as much as thirty per cent. in its composition. This gas becomes more dangerous, not only on account of the large percentage of carbon monoxide, but because of its less disagreeable odor, which renders it more liable to escape detection. The danger of such gas becomes very evident when it is remembered that $\frac{1}{10}$ of one per cent. of carbon monoxide in the air of rooms causes uneasiness and discomfort, while one and two per cent. may result in death. Another factor in the poisonous properties of illuminating gas is the absence of oxygen, which renders it irrespirable.

The physiological action of carbon monoxide has been the subject of much study, but the question of its toxic action upon the tissues remains unsettled. The prevailing view, however, is that it acts directly as a narcotic and depressant upon the nervous system, in addition to the changes it causes in the blood.

Its absorption is rapid, but the amount that the blood retains is not very great. It immediately combines with the hæmoglobin of the blood in the same proportions as oxygen does, displacing that gas, and its combination being more stable it renders the reabsorption of oxygen a difficult matter. The result is that there is "a paralysis of the red blood cells," which no longer absorb oxygen and carry life to the tissues. That there is also a toxic effect upon the nervous system is evidenced by the onset of nervous symptoms before the condition of asphxyia has made any advance, and in many fatal cases a very large proportion of oxygenated hæmoglobin is to be detected in the blood. There is also the evidence of the profound disturbance of the nerve cells in the numerous sequelæ that follow recovery in such cases.

The *symptoms* vary with the amount of poison in the air inspired. When it is weak there are early symptoms of deficient oxygen, during which coma may come on gradually or appear suddenly and become profound. In other cases where there is a sudden overwhelming supply of the poison, insensibility may occur so rapidly that escape cannot be made into purer air. The course in a great measure is influenced by the same conditions. When the onset is gradual the coma is more likely to be deep and to last for days, while in the case of sudden onset, if the subject is quickly resuscitated, the effects pass off more rapidly. When the gas is first inspired there is a sense of discomfort, with fulness of the vessels, pain in the temples, headache which may become very severe and persistent, burning of the skin and face, dizziness, muscular debility, and prostration. Nausea and vomiting are frequent and convulsions may occur. Preceding the onset of insensibility there may be confusion of ideas, disturbed vision, and somnolence or delirium. There may arise a redness of the surface due to vaso-motor paralysis, which may be general or localized. The pulse is generally full and bounding and rapid. The respirations are accelerated and labored and dyspnœa may be severe. The skin becomes dusky, with lips and extremities blue. During the comatose state the limbs and muscles may remain rigid, and convulsions recur frequently. Elevation of temperature is present throughout. Toward the close complete prostration may take place and death follow from asphyxia. When recovery takes place convalescence is slow and numerous complications may persist. The insensibility or dulness of intellect, with loss of memory, may continue after respiration has been established and the condition of the blood has become normal. Convulsions, spasms, and various forms of paralysis may also continue for days after all carbonic monoxide has been removed from the blood. Pneumonia and bronchitis may be caused, albuminuria and diabetes also may persist.

The course of the poisoning is slow, unless the gas has been inhaled in a concentrated form for some time. In such cases death may follow in a few hours, but more generally life remains for some days. The fatal quantity cannot be determined, the injurious effects depending more upon the amount of the poison in the air than upon the quantity of the poison inhaled, as a small percentage may be respired for days with hardly any effect, whilst a slightly greater degree of concentration will produce all the severe toxic symptoms in a short time. The probability of recovery diminishes with the length of time the patient has been exposed to the effects of the poison. It has been estimated from experimental work and clinical evidence that if eight hours have elapsed and the coma is profound there is very slight hope of recovery, but if the time of exposure to the poison has been less the prognosis is more favorable.

The blood in the veins and arteries becomes of a bright cherry color, which is retained for some time after death, and when shaken forms a violet froth which is very distinctive. This is due to the presence of the carbon monoxide, to detect which the spectroscope becomes of service. The test is based upon the fact that hæmoglobin in combination with carbon monoxide is not changed by the addition of reducing agents as is the oxyhæmoglobin of ordinary blood. When viewed through the spectroscope the blood in both conditions shows two absorption bands, which vary a little in position, and by experts the difference is easily detected. But a very marked change takes place when a reducing agent is added to the two solutions: in the one no alteration is affected, in the other the two bands disappear and a single broad band of reduced hæmoglobin takes its place. This is the most trustworthy test for the gas, and it may be of value as a means of diagnosis before death as well as after; but it is not always applicable as the reaction does not take place if there is less than twenty-seven per cent. of the hæmoglobin saturated with the gas (*Johns Hopkins Hospital Bulletin*, December, 1893).

The following abstract of a very valuable report of a case given in detail by Dr. Hewetson is typical of acute poisoning:

A man, 47 years old, was exposed to escaping gas in a trench for less than three minutes, from which he was removed insensible. Three hours later he was unconscious, respirations 40, not shallow; pulse 140, full and of high tension; temperature, 100°. The face was cyanosed, eyes rolling, pupils equal, somewhat dilated, reacting to light, corneal reflex present. The muscles were in a state of tetanic spasm, the limbs were extended and stiff; the deep reflexes were increased. Urine and fæces had been passed involuntarily. The blood showed no change beyond a moderate decrease of the hæmoglobin; $\frac{1}{300}$ grain nitroglycerin was given. The second day the condition was much the same, temperature 101°. On the third day the man had two convulsions which were repeated on the fourth and fifth days. In the intervals the patient remained in the same state. The urine contained no albumin and no sugar. On the sixth day coma and general stasis continued, but the reflexes began to disappear; temperature 103°, respirations 38, pulse 96. The chest sounds were clear. The blood gave no evidence of the poisonous gas when examined with the spectroscope. On the seventh day in the evening and on the following day he had several convulsions and showed signs of depression; pulse 108, low tension; respirations 48 with expiratory groan and gurgle. Two bright-red spots were seen over the left eyebrow. Toward evening he became much weaker; pulse 120–130, respirations 60. Bubbling râles were heard on auscultation and there was deficient resonance. Strychnine $\frac{1}{30}$ grain was given and repeated. The following day he was very low, face bathed in perspiration; respirations 60, pulse 99, temperature 102° to 104°. The arms and legs were rigidly extended, patella and ankle clonus marked. The respiration and pulse grew weaker and death took place in the evening. Post-mortem examination negative (*Johns Hopkins Hospital Bulletin*, December, 1893).

Treatment.—In the management these cases the patient should at once be removed into pure air and measures adopted to expel the poison from the blood. If death has not taken place early, the treatment should be persisted in hopefully as the recuperative power of the blood causes the gas to be slowly expelled, and there is a natural tendency to recovery. This natural curative process is to be assisted and hastened by promoting the natural interchange of gases in the lungs. The removal of the patient into a pure atmosphere may be sufficient, but in severe cases the depression is so great that the respiration must be assisted. Artificial respiration, the faradic current to the phrenic nerve, cold affusions, and heat to the surface and extremities are called for. If these are not sufficient, inhalations of oxygen should be employed, which have acted with very decided benefit in many cases. In extreme cases the transfusion of healthy blood and normal saline solution have been adopted with success. No drug has been suggested as an antidote, although nitrite of amyl and nitroglycerin are frequently employed. The peroxide of hydrogen might supply some of its oxygen to the system and prove of benefit. The various conditions resulting from the impure blood and the action of the poison upon the nervous system will each require its special treatment, which must of necessity vary with the individual case.

POISONING

FROM

LEAD, ARSENIC, ZINC, COPPER, MERCURY, SILVER, AND PHOSPHORUS.

BY

JAMES STEWART,

MONTREAL.

POISONING

FROM

LEAD, ARSENIC, ZINC, COPPER, MERCURY, SILVER, AND PHOSPHORUS.

LEAD.

ETIOLOGY.

It is well known that there is a great variability in the susceptibility of different persons to the poisonous influence of lead. Given a certain number who are exposed to the same influence, it will be found that a few are profoundly affected, some only slightly, while others escape entirely. Persons with an hereditary tendency to gout are very liable to suffer if exposed to the action of lead. Women suffer earlier and more markedly than men when exposed to the same influence. This susceptibility was strikingly exemplified in one thousand cases of lead-poisoning which occurred in Yorkshire from drinking water contaminated by the metal. It was found that the proportion of women to men was as 4 to 1.

Lead-poisoning is met with at all ages. It is more frequent in adult life on account of the much larger number exposed at this period.

The sources of lead poisoning are very numerous. They may be classified into two groups: the industrial and the accidental. The industrial group comprises poisoning from the manufacture of lead in its various forms; the preparation of white lead is especially dangerous, and the manufacture of lead in other forms for lead paint is also a frequent source of poisoning.

Those whose occupation necessitates the employment of lead, especially in the form of the carbonate, are frequent sufferers from poisoning.

Painters, plumbers, type founders and setters, pottery glazers, leather cutters, glass grinders, shot-makers, seamstresses who use thread dyed with lead chromate, workers in bottling establishments who use shot in cleaning bottles, are all engaged in work which exposes them to lead-poisoning.

The accidental sources of lead-poisoning are very numerous. It is often difficult to determine the source of the poisoning in certain cases. We have frequently met with cases of lead-poisoning in women when the source of the lead impregnation could not be ascertained even after prolonged and diligent inquiry.

The most frequent source of accidental lead-poisoning is contamination of water and food.

The contamination of drinking-water by leaden pipes or by lead-lined cisterns is in some parts still a not infrequent event. It is well known that certain waters act on lead while others do not.

Within the past years several epidemics of lead-poisoning have occurred in towns in the north of England due to the contamination of the drinking-water with lead in consequence of the formation of an acid from vegetable decomposition. This is apt to occur in peaty soils. The coating of insoluble salts on the inner surface of lead pipes is considered to be to a certain extent protective, but if vegetable acids form to any-considerable extent in the water supply such protection is not wholly efficient.

Next in importance to water contamination as a source of lead-poisoning is the adulteration of various food-stuffs with lead in some form, especially with the lead chromate which is sometimes used as a coloring matter to color sweetmeats, butter, etc. Dr. David D. Stewart, of Philadelphia, in a very valuable paper has traced no less than sixty-four cases of lead-poisoning to this source.

In the year 1866, a large number of cases of lead-poisoning occurred in Orange County in the State of New York, from the use of flour contaminated by lead which had been used to fill the crevices in the mill-stones. Numerous cases have also been recorded of poisoning from soda water which had become contaminated with lead as a result of the use of patent stoppers containing this mineral.

Infants have been poisoned by lead contained in the rubber nipples of nursing bottles.

Carbonate of lead is sometimes found as an ingredient of certain cosmetics, and a few cases of poisoning have been reported from this source. The use of a lead hair-dye has also caused lead-poisoning.

Comparatively few cases of lead-poisoning are reported from the internal or external use of lead for medicinal purposes.

Rare cases of lead-poisoning have occurred from the inhalation of lead particles while sleeping in newly painted rooms, or from the use of tobacco or snuff which has been kept in lead foil.

The Absorption of Lead.

Lead enters the blood from the alimentary canal, through the skin, and by the way of the respiratory tract.

It is chiefly through the alimentary tract that poisoning takes place. It is in this way that painters and most other workers in lead absorb the metal.

Lead is stored up in the various organs for an indefinite length of time. After further absorption is prevented it is many months and not infrequently years before it is got rid of. It is deposited in the liver, bones, kidneys, muscles, central and peripheral nervous system. In different cases the seat of the chief deposition varies; at one time being most abundant in the nervous system, and again in the muscles or liver.

Lead is eliminated chiefly with the urine and to a slighter extent with the bile and saliva. The skin takes but an insignificant part in this process.

SYMPTOMS.

The chief symptoms in cases of chronic lead poisoning are brought about by the disturbances of nutrition, and of the digestive and nervous systems.

Usually the first, and in severe cases a very marked, symptom is *anæmia*. The pallor more closely resembles that seen in pernicious anæmia than in the ordinary symptomatic anæmias or chlorosis. A further resemblance is that the hæmoglobin is reduced in proportion to the corpuscles, but the corpuscular reduction very rarely reaches that extreme degree met with in cases of pernicious anæmia. It is uncommon to find a greater loss than fifty per cent. in lead anæmia, while in pernicious anæmia reductions of seventy-five to eighty per cent. are the rule even in comparatively early stages of the disease. The loss of hæmoglobin seldom exceeds fifty per cent. in lead poisoning.

There is no change in the absolute number of the leucocytes. The anæmia of lead is due to the interference with the blood function of the bone marrow.

The general nutrition suffers partly as a result of the anæmia and partly from the disturbance of the functions of the gastro-intestinal tract and kidneys.

It is not uncommon to meet with a slight degree of pyrexia even in very chronic cases, but the rule is to find no elevation of temperature. Hyperpyrexia has been noted in a few cases of convulsions and coma due to lead-poisoning.

A characteristic sign of lead-poisoning is the presence of a *bluish line* on the gums. It is met with in all cases with very few exceptions, these latter being usually persons with clean, well-preserved teeth. It may be present over the roots of all the teeth or of only one. It is generally most marked over those of the lower jaw, especially the canines. It is due to the deposition of lead sulphide outside of the vessels. The presence of sulphur is accounted for by the decomposition of albuminous matter in the mouth. Bluish patches varying in size from a minute point to an inch in length are now and then met with in addition to the blue line on the gums.

In one case observed in a woman who had the habit of keeping strands of thread colored with lead chromate in her mouth, a bluish patch of more than an inch in length and half an inch in diameter was present in the mucous membrane of the lower lip. This patient presented all the other common symptoms of lead poisoning.

Gastro-Intestinal Symptoms.—Among the earliest symptoms of chronic lead-poisoning we must place severe paroxysmal abdominal pain, "lead colic." It is generally referred to the region of the umbilicus, coming on in paroxysms of varying duration, from minutes to hours or even several days. The pain is usually of an intense character and relieved by pressure. In the intervals between the paroxysms relief may be complete or incomplete, when incomplete a dull "wearing" pain being complained of by many patients.

During the paroxysms of abdominal pain, the abdomen is retracted and vomiting often takes place. Obstinate constipation is a prominent symptom of chronic lead-poisoning. It is especially intense during the days when the abdominal pain is present.

The pain and the constipation are probably due to the same cause, the tetanoid contraction of the intestinal muscles. It is a disputed point as regards the mode of causation of this tetanoid contraction, whether it is brought about by a direct irritant action of the lead on the muscular structure of the intestine or through the nervous mechanism. This point will be further dealt with when the treatment of the condition is under consideration.

Attacks of pain of a paroxysmal character may be referred to the epigastrium. Such attacks may alternate with abdominal colic or they may be the only symptom referable to the gastro-intestinal tract that is complained of.

Arterial and Renal Symptoms.—During the attack of intestinal colic, the pulse is slow and hard, and the vessels are tense. The aortic second sound is accentuated. All of these states pass away when the intestinal symptoms (colic) subside. There comes a time in many cases of chronic poisoning, however, when the arterial tension is permanently raised and the heart is hypertrophied. This is due to either a general arterio-sclerosis or an arterio-sclerotic nephritis, or both states combined.

Recently we have seen a case of general arterio-sclerosis with interstitial nephritis in a painter, twenty-eight years of age, who first presented symptoms of lead-poisoning (colic) two years previously to coming under observation. The general arterio-sclerosis in this case had reached a very high degree. The degree of frequency of arterio-sclerotic changes in chronic lead-poisoning is a matter on which various opinions are expressed. In cases that have lasted more than a year, it is in our experience an almost constant condition, although differing greatly in degree in different cases.

At times the arteries appear, as it were, to bear the brunt of the poisoning, while in other cases the changes in the vessels are comparatively slight. Arterial changes are in some respects the most important that attend lead-poisoning. They indicate irretrievable damage, not unfrequently leading to secondary changes in the kidneys and to rupture of cerebral arteries.

The arterio-sclerosis, instead of being a primary and direct effect of lead, may be induced secondarily through the kidney changes, the latter being brought about by the irritating action of the mineral.

Disease of the kidney may be either parenchymatous or interstitial, the former being in all probability a direct result of the irritant while the latter is indirectly induced through the arterial changes. Parenchymatous nephritis in its advanced stages is usually accompanied with interstitial changes.

The action of lead on metabolism will be appropriately considered in this connection. It is generally agreed that there is a deficiency of both urea and uric acid in cases of advanced lead-poisoning, but it is a curious fact that the frequency of so-called saturnine gout varies much in different places.

Alfred Garrod, Haig, Duckworth, and others practising in London consider that the association of lead-poisoning and gout are very frequent, while Oliver, practising in Newcastle, meets with gout comparatively rarely as an effect of lead. Gout appears to be a very uncommon occurrence in cases of lead-poisoning in America. We have noticed, since our attention has been drawn to the matter through a perusal of Oliver's work, that those who have been great beer drinkers are more apt to develop symptoms of gout than others. In the south of England, beer is the favorite beverage of workmen, and to this custom is attributed the frequency of gout in lead-poisoning.

It is questionable whether lead induces a true gout or not. Sir William Roberts is of the opinion that uratosis is a manifestation of different factors. He says: "The gouty diathesis and lead-poisoning, while differing in all other respects, have one tendency or vice in common, viz., the tendency to uratosis."

Nervous Symptoms.—The most frequent nervous symptom due to lead-poisoning is bilateral paralysis of the extensors of the wrist and first phalanges, producing double "wrist-drop."

The paralysis, although nearly always bilateral, is seldom equally marked in both hands, the right arm commonly being affected first and to a greater extent than the left. The onset of the paralysis is usually slow, two or three weeks generally elapsing between the first appearance of any weakness and complete loss of power. In the majority of cases it goes on to a complete loss of power. It is much more common in second than in first attacks of saturnism.

The loss of power is generally first noticed in the index finger. It soon (in a few days) extends and involves all the extensors of the first phalanges including the thumb, as well as those of the wrist.

The muscles involved are those innervated by the musculo-spiral nerve. There are, however, three muscles supplied by this nerve which nearly always escape; these are the triceps, the long supinator, and the extensor of the metacarpal bone of the thumb. It will therefore be seen that certain special muscular groups are picked out.

Now and then we meet with cases where the paralysis is more marked in the upper than in the lower arm groups. The deltoid or the biceps may suffer more than any of the others.

Sometimes the muscles innervated by the peroneal nerve are paralyzed. The flexors of the hands escape, although their usefulness is much interfered with through the loss of power in their antagonists. The paralyzed muscles undergo atrophy. Lead paralysis is therefore an atrophic paralysis. The reaction of degeneration is present except in the slighter cases, the reaction of the muscles to faradism being *nil*, while to galvanism we get the usual slow but voluminous reaction. In severe cases this latter reaction will also pass away in the course of a few months. The normal electrical formula is changed, A O Z < K S Z (An O C < Ka C C) being the formula for the reaction in the completely paralyzed muscles. In certain muscles which appear to act normally to the influence of the will it will often be found that the faradic reaction is diminished and the galvanic increased, but we never meet with the full reaction of degeneration in non-paralyzed muscles.

There is a second form of lead paralysis, affecting also for the most part the small muscles of the hands and characterized chiefly by wasting, the paralysis depending on the amount of wasting. Here the atrophy is primary. Both forms of paralysis are commonly found together, it being rare to meet with a case of pure primary muscular atrophy. The disease closely resembles progressive muscular atrophy, the electrical reactions being the same in both cases, the loss of reaction depending on the extent of atrophy. Even in the severest cases it is possible to obtain some reaction to the faradic current.

As pointed out by Gowers, the primary atrophic form of lead paralysis is much more obstinate than the degenerative form; it is not uncommon to meet with it as a permanent condition.

General paralysis has been described as the result of lead-poisoning, the muscles of the extremities and trunk and the intercostals being all involved in this unusual form. Recovery has followed in some instances, but it can be readily understood how serious such cases are, and how death may supervene from respiratory paralysis.

Tremor is not an infrequent symptom of lead-poisoning in very chronic cases. It is, however, not so common in lead as in mercurial paralysis. It may be fine or coarse.

Considerable sensory disturbances are rarely met with in any of the forms of lead paralysis. Pain may be complained of, but it rarely is localized to the course of a nerve trunk. I have generally found it confined to the joints (lead arthralgia).

A slight diminution in tactile sensibility may be made out on careful examination in the region of distribution of the musculo-spiral nerve. Marked hemianæsthesia involving the extremities only may be met with in cases of lead-poisoning, but it is doubtful whether such cases have any direct connection with the lead-poisoning. It is well known that lead may induce hysteria in either sex, and it is highly probable that the hemianæsthesia is functional and not organic—hysterical and not saturnine. The same explanation holds as regards the origin of a "glove" anæsthesia of both hands, which has been described as occurring in chronic lead-poisoning.

The term "*lead encephalopathy*" is used to describe certain cerebral manifestations of lead. These symptoms include convulsions, delirium, coma, aphasia, hemiplegia, hemianæsthesia, amaurosis, hemianopsia, hysteria, insanity, etc.

Convulsions of various types are the most frequent, and have a special interest on account of their liability to be mistaken for convulsive movements due to other causes. In a recent paper by Dr. D. D. Stewart, of Philadelphia, founded on sixteen cases, we have a very complete description of the different varieties of convulsive movement from lead-poisoning. In one of Dr. Stewart's cases, the convulsions appeared as early as the fifteenth day after exposure; in thirty-two days in a second. In one case there was an interval of twenty years between the appearance of the convulsions and the exposure. We have seen convulsions appear in the fourth month in one case and in the ninth month in another case. It is generally conceded that convulsions and other severe cerebral symptoms occur only in those who are engaged in the manufacture of lead. This is true, however, only of the majority of cases.

The types of convulsive movements met with are:

1. Severe and repeated convulsions lasting from a few hours to days and nearly always terminating fatally;
2. Convulsive movements which clinically resemble in their character and recurrence those of idiopathic epilepsy;
3. Movements which may resemble ordinary general convulsions of an hysterical or hystero-epileptic character.

The prognosis in cases of either of the two latter types is on the whole very favorable, provided their nature is recognized and proper treatment instituted.

Delirium of an acute restless character is met with, but is a rarer symptom than convulsions. Lead delirium may closely simulate the acute delirium of alcohol, being accompanied with special sense hallucinations and muscular tremors.

Coma as the result of lead-poisoning is a very grave symptom. It is generally preceded by convulsions, or the condition may be a true status epilepticus—recurring convulsions in a comatose state.

Lead insanity is comparatively rare. It may take the form of melancholia, alcoholic insanity, or general progressive paralysis.

Lead-poisoning in young girls is often the exciting cause of repeated hysterical outbreaks. Hemianæsthesia with hemiplegia occurring in a person suffering from lead-poisoning may be due to:

1. Functional disturbance (hysteria);
2. Hemorrhage into or softening of the internal capsule (lead arterio-sclerosis);
3. Uræmic poisoning.

Transient hemianopsia, or amaurosis, is also occasionally met with. Optic neuritis is a frequent symptom in lead-poisoning attended by the severer cerebral disturbances. It may pass away or go on to pronounced atrophy of the discs. Color vision may be greatly limited in these cases. Paralysis of the laryngeal and external ocular muscles are among the rare effects of lead-poisoning.

Pathological Anatomy and Pathology.

The chief lesions occurring as the result of the action of lead are to be found in the central and peripheral nervous systems, muscles, mucous membranes, liver, and kidneys.

The wasted muscles are found of a pale or pale yellow color. The change in color becomes more marked as the wasting advances. It is generally found that the wasting is of a simple character, not being attended by any fatty or pigmentary changes. This primary atrophy is accompanied by a multiplication of the nuclei of the sarcolemma. In advanced cases the sheaths of the sarcolemma become destitute of muscular fibres altogether, connective tissue taking their place. Side by side we may find apparently normal fibres and those which have undergone almost complete atrophy.

The vessels in the muscles are usually found in a state of arteriosclerosis.

The nerves in the paralyzed parts are always found to have undergone changes more marked in their peripheral terminations in the paralyzed muscles than elsewhere. The chief nerve trunks of a paralyzed limb also present evidence of degenerative changes, grayish-white patches being found in the musculo-spiral, resembling the sclerotic patches met with in cases of disseminated sclerosis of the central nervous system.

The changes met with in the nerves ending in the muscles resemble closely that described as "Wallerian" degeneration following an injury to a nerve, constituting a parenchymatous neuritis. There is segmentation of the myelin and breaking up of the axis cylinder, proliferation of the nuclei of the sheath of Schwann and neurilemma.

In the early stages and in slight cases the changes are less marked; they are limited to the medullary sheath, constituting what Gombault has termed periaxial neuritis. These neuritic changes he found in segments, diseased tracts being separated by healthy parts, "segmental neuritis." In the course of the musculo-spiral nerve he frequently found the profound parenchymatous and the periaxial neuritis together, the former in the muscular twigs, the latter in the main nerve trunk and its larger branches. The changes are constantly present in the nerve terminations in the muscles, and gradually diminish in intensity as we travel to the cord. Slighter changes are met with in the anterior roots in some cases. In five cases examined by M. Déjerine he found neuritic changes in the anterior roots in two cases only.

In the majority of cases the cord has been found healthy. In a few (Oppenheim and Viller) there was some wasting of the cells of the anterior horns. These changes are, however, very inconstant.

There are no constant macro- or microscopic changes met with in the brain to explain the cerebral symptoms of lead-poisoning. Slight lepto- and pachymeningitis have been found. In both the cord and brain vascular changes are frequent, and as a direct consequence of these, interstitial tissue increase is found. Patches of softening and capillary or even more extensive hemorrhages may also result from the arterio-sclerotic changes.

Oliver describes the changes met with in the liver as the result of lead-poisoning. He found atrophy of the hepatic cells and increase of connective tissue, changes closely resembling syphilitic hepatic cirrhosis. The changes met with in the kidneys are indicative of interstitial and parenchymatous changes.

For many years it was a matter of doubt whether the cause of lead paralysis resided in central or peripheral changes. The careful researches of Gombault referred to have conclusively settled this question. There can now be no doubt that the paralysis is due to a peripheral neuritis. This neuritis is due to a degenerative process. The reason why the sensory fibres usually escape, and why the process is usually confined to the terminations of the musculo-spiral nerve, remains unexplained.

Gowers ascribes the intense primary atrophy of lead to degeneration of the ganglion cells of the anterior horns.

Lead encephalopathy must be due to the functional disturbance of the cerebrum from the deposition of lead in it, or to the retention of poisons from renal and hepatic inadequacy, for it has been pointed out that macro- or microscopic changes are absent; the only exception to this being the lepto- and pachymeningitic changes which have been seen in cases having the clinical course of a progressive general paralysis. The muscular degeneration in lead-poisoning is clearly a secondary process, brought about by the degenerative neuritis.

DIAGNOSIS.

There is seldom any difficulty in the diagnosis of lead-poisoning. In the great majority of cases we have present several of the most characteristic symptoms. These are: (1) the intestinal colic; (2) the anæmia; (3) the blue line on the gums; (4) the bilateral musculo-spiral paralysis.

When the above symptoms are met with in a person who has been exposed to lead-poisoning in any form the diagnosis is absolutely certain.

Very rarely the paralysis of alcoholic neuritis may resemble that of lead. In alcoholic paralysis, even if it be confined to the muscles innervated by the musculo-spiral, the sensory symptoms are usually a leading feature, especially extreme tenderness of the muscles. In arsenical neuritis, the lower extremities suffer equally with the upper, and sensory disturbance is here also a marked symptom.

There may be a difficulty in diagnosis between lead-poisoning and chronic poliomyelitis, when the characteristic symptoms of the former, such as blue line and colic, are absent. In such cases an examination of the urine should be made. There may also be a difficulty in the diagnosis of lead epilepsy from idiopathic epilepsy. The urinary examination will make the nature of the cause clear.

Dr. J. J. Putnam, of Boston, in his article on lead-poisoning in Keating's "Cyclopædia of the Diseases of Children," gives the following as the method recommended by Dr. A. M. Comey of Harvard University for the detection of lead in the urine:

"The urine is first evaporated to dryness, then fused in a crucible, with the addition of a little pure nitre, till it becomes white. The crucible is then cooled and dilute hydrochloric acid added, hot, to extract the residue after ignition. It is then filtered, and the filtrate treated with ammonia to alkaline reaction to precipitate the phosphates and iron. Sulphide of ammonium is added at the same time, which throws down the sulphide of iron and lead. This is washed three times by decantation with hot water, then water is added, and the whole is acidified with hydrochloric acid, and allowed to stand until the next day. It is then filtered through a small filter and the residue washed. A little pure (free from iron) nitric acid is then added, drop by drop, by which the sulphide of lead, if present, is dissolved and carried through as nitrate of lead. This is collected in a watch-glass, evaporated to dryness, and the final test made by the addition of a drop of water and a crystal of iodide of potassium. Finally, to eliminate bismuth, the iodide of lead is again dissolved in hot water, filtered, and reprecipitated in a perfectly clean test-tube with dilute sulphuric acid. The test-tube is allowed to stand twenty-four hours, and is then twirled gently between the fingers. The sulphate of lead, if present, rises in a delicate spiral."

PROGNOSIS.

The prognosis in the milder manifestations of lead-poisoning is good as regards not only danger to life but recovery from the effects. In the lighter degrees of paralysis of the musculo-spiral the power is nearly always regained after a few months. Where the paralysis is profound and the reaction of degeneration present, recovery nearly always occurs, although many months or even a year or two may elapse before it is complete. The prognosis in primary atrophy (chronic lead poliomyelitis) is much less favorable, complete recovery seldom occurring, but the condition is not progressive if all the lead has been eliminated. Hence there is no direct danger to life, the paralysis in this respect being different from chronic progressive muscular atrophy of myelopathic origin.

The prognosis in the relapsing cases is always less favorable. It is, however, not uncommon to meet with cases where three or even four recurring attacks of lead paralysis have been completely recovered from. The prognosis is much graver when the severer cerebral symptoms of poisoning are present.

Coma and repeated convulsive movements are always extremely serious. The same is true of chronic mental disturbance, whether it takes the clinical form of melancholia or of progressive general paralysis.

The ultimate prognosis in all cases depends on the integrity of the arteries and kidneys.

TREATMENT.

This will be considered under the following heads:

1. The preventive treatment.
2. The promotion of the elimination of the poison.
3. The treatment of the symptoms.

Preventive Treatment.

If due and proper precautions are taken by artisans engaged in dangerous occupations it is quite possible in many cases to prevent the ingestion of the poison. There is no doubt that people vary greatly as to their liability to suffer from lead-poisoning. With the exception of lead-workers, the people in this country who are most frequently the victims of poisoning are painters. There can be no doubt that the danger of poisoning in this trade can be much lessened by proper precautions. Thorough cleaning of the face and hands previous to eating will greatly lessen the danger. We have been told by an old painter, who in his early years had lead colic, that he was able by strict cleanliness to save himself from any such danger. It is his habit not only to be cleanly himself but to teach cleanliness to his fellow-workmen. He considers that he has saved many a poor man from becoming poisoned by lead.

Much yet remains to be learned about the various ways in which lead may find entrance into the system. In several instances occurring in women, we have been unable to trace the source of the lead-poisoning. In such cases it will be necessary to give directions about the drinking-water, the use of cosmetics, of colored thread, and the various other possible sources of lead. In all lead factories conducted by intelligent and right-thinking people every precaution is taken to lessen the risks of the work-people. The use of sulphuric acid or of magnesium sulphate and sulphuric acid has for a long period enjoyed a reputation as a preventive.

Oliver lays particular stress on the necessity of every man and woman having a substantial meal before beginning the day's work, and of the prime necessity of insisting on total abstinence.

Promotion of the Elimination of Lead.

Lead is eliminated chiefly by the kidneys and intestines. Iodide of potassium is our chief agent for the getting rid of lead from the tissues. How it acts is unknown, though probably it is through some influence on metabolism. The dose of the iodide should not as a rule exceed fifteen grains twice in the twenty-four hours, as it is commonly taught that large quantities may throw a sufficient amount of lead back into the blood to bring about acute poisoning. In chronic cases, especially in old paralytic cases, such a danger is very remote. Any such danger can be prevented by the use two or three times a week of a saline purgative, preferably the sulphate of magnesium.

By the constant use of iodide of potassium and the occasional employment of sulphate of magnesium we are promoting the elimination by the two chief channels through which lead leaves the system.

Symptomatic Treatment.

It is necessary to use morphine for the relief of intestinal colic, when severe, as it usually is. This agent not only relieves the pain, but relaxes the tetanoid spasm on which the constipation depends.

Iron should be given when anæmia is present. For the paralysis, galvanism should be employed daily, the anode to be placed over the sternum, and the cathode to be moved along the paralyzed muscles. The nutrition of the muscles will in this way be kept up, and the recovery will in consequence be earlier and more complete than it otherwise would be. There is no special treatment for the cerebral symptoms. The principles governing the treatment of the arterial and renal changes remain the same whether these changes be due to lead or to other causes.

ARSENIC.

Acute Poisoning.

ETIOLOGY.

The sources of acute arsenical poisoning are very numerous. The effects depend upon the oxygen compounds, arsenious and arsenic acids. Both of these acids cause in man and mammals violent gastro-intestinal symptoms. No matter how arsenic may be introduced it gives rise to symptoms of gastro-intestinal irritation. As they appear a few minutes after the introduction of the agent and before its slowly acting acid properties could take effect, it is much more probable that the vomiting and purging are due to the action on the circulation than to a direct local action on the gastro-intestinal mucous membrane.

There is great depression of the arterial blood pressure, more particularly of that of the abdomen.

The symptoms of poisoning usually make their appearance from fifteen to thirty minutes after the introduction of the metal.

Severe burning pain in the abdomen of a cramp-like character is first complained of in the great majority of cases. This is soon followed by vomiting and purging. In a small percentage of cases the abdominal symptoms may be slight or entirely wanting. The clinical picture often closely resembles that of Asiatic cholera, viz., copious rice-water stools with extreme prostration.

The cases of acute arsenical poisoning unattended by gastro-intestinal symptoms are characterized by disturbance of the functions of the central nervous system, the so-called "arsenicismus cerebrospinalis." In such forms we meet with giddiness, delirium, convulsions, paralysis, and coma.

The symptoms again may be entirely those of a profound prostration, there being an absence of gastro-intestinal and nervous symptoms.

In the nervous variety of poisoning, death quickly follows. It is highly probable that cases characterized mainly by nervous symptoms and by prostration are due primarily to the action of the metal on the circulation, brought about by the great reduction in the blood pressure.

There is probably no direct action on the nervous system in cases of acute arsenical poisoning.

PATHOLOGICAL ANATOMY AND PATHOLOGY.

In the stomach and intestines hemorrhages into and on the mucous membrane are frequently found. The epithelium is degenerated and detached. Peyer's patches and the solitary glands are swollen. Patches of ulceration are also frequently present. Parenchymatous and fatty degeneration of the liver, of the epithelium of the urinary tubules, of the heart and voluntary muscles are constantly to be observed if the patient has lived twenty-four hours or longer.

TREATMENT.

The treatment of acute arsenical poisoning consists in first neutralizing and getting rid of the poison in the stomach and intestines. A freshly prepared sesqui-oxide of iron, made by neutralizing the tincture of the perchloride of iron with sodium carbonate, is generally employed. Dialyzed iron may also be used, but neither of these iron preparations is effective unless the arsenic is in solution. The stomach should be thoroughly washed out by means of the stomach-pump or by emetics.

The symptomatic indications should be met on general physiological principles.

Chronic Poisoning.

ETIOLOGY.

The sources of chronic arsenical poisoning are: (1) the use of arsenic in medicinal quantities for long periods; (2) the ingestion of a single poisonous dose, either from accident or design; (3) the ingestion of arsenic from wall-papers, carpets, etc., containing it.

Considering the extensive employment of arsenic in various diseases, it is surprising how comparatively rarely pronounced symptoms of poisoning from it are seen. We exclude the milder acute manifestation of irritation of the mucous membranes.

The shepherds of the Styrian Alps, who are accustomed to take arsenic continuously for years, extremely rarely suffer from either acute or chronic disturbance as the result of their habit. Very many cases have, however, been recorded of pronounced chronic symptoms resulting from the use of even small medicinal doses for comparatively short periods.

Putnam, Da Costa, Osler, and many others have published examples of grave poisoning (paralysis, etc.) resulting from the medicinal use of moderate doses of arsenic.

Symptoms of acute and chronic poisoning as the result of single large doses are very numerous.

Chronic arsenical poisoning from the inhalation of pigments used in the manufacture of various domestic articles is now universally recognized as being more frequent than was formerly supposed.

J. J. Putnam, of Boston, has through his writings made this clear. The following materials sometimes contain arsenic in quantities that may prove dangerous: various kinds of wall-paper, carpets, labels, colored papers used for different purposes, wax ornaments, and colored toys.

The Elimination of Arsenic.

Arsenic is chiefly eliminated by the kidneys, but in very chronic cases it may be found in all the secretions and excretions.

Like mercury and lead it may be found in the urine after many months.

Symptoms.

The symptoms of chronic arsenical poisoning are essentially those due to a disturbance or loss of function in the peripheral nerves.

The usual minor symptoms met with in cases of a slight overdose, such as irritation of the conjunctiva and gastro-intestinal tract, may precede the nervous symptoms. In the developed cases, however, it is the rule to find the former absent.

Arsenic as far as is known does not appear to influence the general nutrition to the same extent as does lead. In the few cases that we have seen anæmia was not present.

Dr. Putnam refers to an increase of weight from the absorption of arsenic from wall-papers.

Arsenic seriously interferes with the complete elaboration of the changes that take place in the albuminous tissues. It prevents the fats from undergoing transformation into carbonic acid and water; hence the fatty degeneration of the muscular and epithelial structures.

The effect of arsenic in producing cutaneous changes (herpes, pemphigus, pigmentation, etc.) is a proof of its influence on nutrition.

The distinctly nervous symptoms met with in chronic arsenical poisoning may be for clinical purposes arranged in three groups. We have (1) cases where the symptoms are chiefly if not wholly due to partial or complete loss of function in certain motor nerves; (2) those in which the sensory function is the chief or sole disturbance; (3) those in which the symptoms are mainly ataxic—the so-called "pseudo-tabes." Generally we meet with a combined motor and sensory paralysis.

One single overdose of arsenic may bring on any of the symptoms; the intervals between the taking of the poison and the development of the nervous symptoms may vary from a few days to a few weeks. In one case recently observed in a middle-aged woman, the symptoms of paralysis of the lower limbs first showed themselves three weeks after a poisonous dose of Paris green had been taken. This was the second attempt made by this woman to kill herself by the same agent. There were no nervous symptoms following the first attempt. There was an interval of six months between the two attempts at poisoning.

The paralysis due to arsenic, unlike lead paralysis, is not as a rule confined to the extensors of the wrists and first phalanges, but involves the corresponding muscular groups of the lower extremities. There is also a great tendency in arsenical paralysis for the upper arm and thigh muscles to become involved. The paralysis in its depth and distribution is therefore more akin to alcoholic than to lead paralysis. The sensory disturbance, which is slight and often absent in lead paralysis, is usually marked in arsenical paralysis. It is the exception to meet with a pure motor paralysis in arsenic, while it is the rule in lead paralysis.

Rapid wasting occurs in arsenical paralysis, and the atrophied muscles present the reaction of degeneration.

Contractures are very apt to occur in the muscles of the lower limbs in the advanced stages. Tremor of the upper extremities is usually present to a greater or less degree. The tremor is fine.

The sensory symptoms may be, as already mentioned, the most obtrusive feature in arsenical poisoning. The subjective sensory symptoms are usually well marked. In the patient referred to, pain and numbness were very marked. Pain in the course of the inflamed nerves and in the skin and muscles is a frequent symptom. The muscles are very painful on pressure. Loss of the various forms of sensation is the rule in advanced severe cases. Loss of sensation is first noticed and is always most marked in the most peripherally situated parts. The ataxic form of arsenical poisoning is met with in many of the cases where there are also motor and sensory disturbances, and occasionally it is seen almost pure. The ataxia may involve the lower or upper extremities or both. It may be so pronounced that the patient is unable either to stand or to walk. In some cases reported it has proved more enduring than any of the other symptoms present. Romberg's symptom is usually met with in cases of ataxic arsenicismus.

The knee-jerk in cases of paralysis of the lower limbs is generally found diminished or altogether absent.

PATHOLOGICAL ANATOMY AND PATHOLOGY.

The chief morbid changes in arsenical poisoning are to be found in the peripheral nerves. They consist in a degenerative neuritis, differing in no essential particulars from those met with as the result of other toxic agents. The changes are, however, not limited always to the peripheral nerves. Henschen has reported a case where he found extensive degenerative changes in the ganglion cells of the anterior horns extending from the cervical to the lumbar regions. Degenerative changes have also been described as occurring in the medulla. Schaffer in experimental arsenical poisoning in rabbits and dogs found changes constantly in the cord.

From these reports it would appear that arsenic like lead causes a degenerative parenchymatous neuritis. Degenerative atrophy of the spinal ganglion cells appears to be a more frequent change in arsenical than in lead-poisoning.

Henschen found degeneration of the postero-internal columns (Goll's) in the case already referred to. This would explain the presence of ataxia. This symptom may, however, be brought about by a neuritis of the sensory muscle nerves, as has been pointed out by Gowers.

DIAGNOSIS.

The diagnosis of subacute or chronic arsenical poisoning presents no special difficulty when the source of the metal has been ascertained. The clinical picture of a fully developed arsenical paralysis is usually so marked so as to enable one at once to differentiate it from lead paralysis. The frequent sensory disturbances, together with the more marked localization of the motor and sensory loss in the lower limbs, are characteristic of arsenical poisoning, while in lead-poisoning the motor defect is chiefly, if not wholly, in the extensors of the wrists and first phalanges. In the latter it is rare to meet with marked sensory symptoms, either subjective or objective.

The differential diagnosis between arsenical and alcoholic paralysis must rest chiefly on the history and the examination of the urine.

PROGNOSIS.

In severe acute poisoning the prognosis is very grave. In chronic arsenical paralysis recovery eventually takes place in the great majority of cases.

Involvement of the diaphragm or intercostal muscles renders the prognosis very doubtful.

TREATMENT.

The treatment of chronic arsenical poisoning is chiefly the treatment of the neuritis, and does not differ from that of lead neuritis. The elimination of the metal should be promoted by the employment of iodide of potassium and purgatives. If the nerves and muscles are very tender and painful, electricity and massage should not be employed until these symptoms have subsided.

If the poisoning has resulted from the inhalation of arsenical dust from wall-papers, the latter should be entirely removed from the walls.

ZINC.

Acute zinc-poisoning is characterized by severe vomiting, the matters vomited containing more or less blood, by severe gastric and abdominal pain, and purging. Some of the salts, especially the chloride, are violent corrosives. We have here not only the irritant action of chlorine, but also the escharotic action of hydrochloric acid. Death may be brought about by the violence of the irritant action on the gastro-intestinal mucous membrane.

All the zinc salts which can be absorbed into the blood induce in large doses, both in man and the lower animals, paralysis of the voluntary muscles and the heart. The doses sufficient to induce these effects are usually enough to bring about quickly a fatal result.

The symptoms preceding death are those due to the depressant action of the poison on the heart. Convulsions and coma ending in death is another mode of termination in fatal cases of zinc-poisoning.

Nothing is known about the effects of long-continued considerable doses of zinc salts. Experiments by Daletski and Pelikan on feeding animals with zinc and copper salts were negative in their results. We are not aware of any changes having been described in the central or peripheral nervous system, or in any of the internal organs in man, from the long-continued use of zinc salts.

The treatment of acute zinc-poisoning is as follows: (1) The stomach should be emptied; (2) milk mixed with sugar and the white of egg should be freely administered; an insoluble albuminate of zinc is formed, upon which the gastric acids have little or no effect; (3) the various indications must be met as they arise; the principal of these is to combat the depressant action on the heart by alcoholic stimulants and strychnine.

COPPER.

Acute Poisoning.

Copper salts in poisonous doses induce the same train of symptoms as those of zinc. They are, in the first place, gastro-intestinal irritants, and, second, they affect the heart and the central nervous system in the same way.

The treatment of acute copper-poisoning is the same as that of zinc.

Chronic Poisoning.

Chronic poisoning from the various copper salts is more common than chronic zinc-poisoning. Cases have been reported of poisoning from the use of copper in colored confectionery; from the use of copper vessels in cooking; from the use of the metal in the manufacture of artificial flowers; from its employment in the making of pickles and preserved fruits to give them a green color; and from the use of green wrappers for farinaceous foods.

Copper has been used to adulterate bread, and cases of poisoning have been reported from this source.

The local application of copper has been the means of causing symptoms of chronic poisoning.

Workmen in copper mines suffer from certain symptoms which have been attributed to copper. It is, however, probable that in many such cases the effects are due rather to arsenic, which is a common constituent of copper ore.

SYMPTOMS.

As copper is eliminated in part by the gastro-intestinal glands and salivary glands as well as by the kidneys, symptoms of irritation and disturbance of the functions of these structures will usually be found. Nausea, vomiting, and diarrhœa are generally present. The patient complains of a coppery taste in the mouth. Colic resembling lead colic has been met with in cases of chronic copper-poisoning. Disturbance of nutrition is also noticed. The patient loses flesh, and becomes anæmic. Jaundice is a symptom, the cause of which has not been satisfactorily explained. The perspiration colors the clothing of a greenish tint. A greenish or a bluish line appears on the edges of the gums.

The hair is said sometimes to turn of a green tint.

We have met with one case of a peripheral neuritis in a man who was working for many months in the Capleton copper mines in Quebec. The symptoms were chiefly sensory and ataxic, and after many months of treatment with iodide of potassium passed away.

TREATMENT.

Removal of the cause is the first essential in the treatment. The internal use of iodide of potassium to promote elimination of the metal fulfils the second indication. The treatment does not differ from that of poisoning by other heavy metals.

MERCURY.

ETIOLOGY.

The chief source of chronic mercurial poisoning is the inhalation of the vapor of the metal or its dust by those engaged in various manufacturing trades, such as the makers of mirrors, barometers, and thermometers. Bird-stuffers, hat makers, and furriers make use of mercury in the preparation of the skins and sometimes suffer from mercurial poisoning.

As mercury is volatilizable at ordinary temperatures, it enters the blood through the lungs. There are numerous cases recorded where it has produced poisoning through absorption by the skin. It may also enter through the digestive tract in consequence of contamination of the food from the want of cleanliness on the part of those engaged in handling it. Cases are also reported where poisoning has resulted from mercury used in dental work.

It is rare to meet with chronic poisoning from the medicinal use of mercury in ordinary doses when given either internally or applied to the skin or used in the form of solutions for the washing of wounds or cavities. A few cases have been recorded of poisoning from vapor baths of calomel—a form of mercurial treatment still used in syphilis.

Persons vary much as to their susceptibility to the action of mercury. Women and children are more likely to suffer than men. No matter in what form or by what channel mercury is introduced into the blood and tissues, it exists there in the form of an albuminate. It is eliminated with extreme slowness. Ludwig found it in the tissues in cases of chronic syphilis, which had been treated by mercury, many years after the use of the drug had been discontinued. It is got rid of chiefly by the kidneys and the gastro-intestinal mucous membrane.

SYMPTOMS.

The onset of the symptoms may present an acute, subacute, or chronic character.

The acute onset is characterized by the rapid appearance of stomatitis, nausea, vomiting, and a disagreeable coppery taste in the mouth. In the subacute and chronic modes of onset the stomatitis, nausea, and vomiting are usually not met with, the first characteristic symptom being commonly *tremor*. It is generally first felt or noticed in the tongue and lips, and gradually spreads thence, involving the entire voluntary muscular system. At first it is only brought out on movement, but when of some standing it is apparent during complete rest. It is the rule to find that the tremor ceases during sleep, but to this many exceptions have been recorded. In consequence of the tremor of the lips and tongue, the speech is altered. The tremor may be either fine or coarse.

Paralysis of various muscles is met with in certain cases of chronic mercurial poisoning. It seldom goes on to complete loss of power. It may affect the muscles of one or all the extremities, or it may be confined to particular muscles, such as those of the larynx, giving rise to aphonia. There is no change in the electrical reaction of nerves and muscles. The paralyzed muscles do not undergo atrophy. Sensory disturbances are present in many cases, and in the extremities are usually of a subjective character. Pains of a neuralgic nature in the joints are also met with.

The tendon reflexes have been found either normal or exaggerated. There is no disturbance of the innervation of the bladder or rectum.

Great irritability and sleeplessness are usually found. Now and then graver mental symptoms are met with, such as hallucinations and acute mania.

Mercury resembles lead in its tendency to develop hysteria in both males and females. Aphasia of a temporary character is one of the infrequent symptoms of mercurial poisoning. It may either be due to the direct action of the metal or may be an hysterical manifestation indirectly brought about. In the same way hemiplegia, hemi-anæsthesia, and disturbances of the functions of the special senses may be induced. Rarely idiocy may be the result of mercurial poisoning.

The effects of mercury on the general nutrition are commonly manifest in very chronic cases, emaciation and anæmia being usually present. The hair falls out, the nails become brittle, and brown pigmentation of the skin, like that met with in Addison's disease, is seen. Albuminuria with general œdema and hemorrhages into the skin and mucous membrane are evidences of an advanced dyscrasia.

PATHOLOGICAL ANATOMY AND PATHOLOGY.

In the human subject no significant changes have been found in either the central or peripheral nervous system. In chronic poisoning experimentally induced in animals changes have been found both in the cord and in the peripheral nerves, the latter consisting essentially in a destructive change in the myelin by which it is reduced to a granular mass in which the intact axis-cylinders float. The changes met with in the cord correspond to a diffuse myelitis.

Judging from the character and course of the nervous symptoms of mercurial poisoning met with in man, it would appear that the most plausible explanation would be a cerebral disturbance from the deposition of the metal in certain cortical areas. The tremor, the paresis, slight sensory disturbance, the absence of wasting in the paretic muscles, the retention of the normal electrical reactions, the increased tendon reflexes, the non-involvement of the bladder and rectum clearly point to a cerebral rather than to a spinal or peripheral change.

DIAGNOSIS.

Given a history of exposure to the action of mercury, there can be no difficulty in arriving at a conclusion as to the nature of a combination of tremor, paresis, and mental irritability. If it is impossible to obtain such a history, then we shall have the difficulty of differentiating between the various forms of disease in which tremor is a leading and characteristic symptom.

In progressive general paralysis we have a somewhat similar symptom group, but in this disease the peculiar mental disturbance so commonly met with is not the same as that generally found in mercurial poisoning. In the latter it is a mental irritability, while in the former the grandiose ideas and loss of memory are the dominant changes. In general paralysis the pupillary changes (myosis) are nearly always to be found, while they are absent in chronic mercurial poisoning. In the latter the tremor is soon general, while in the former it is confined to the lips, tongue, and hands. In any case the diagnosis will soon be cleared up as the disease progresses.

In the early stages of typical disseminated cerebro-spinal sclerosis the tremor is the predominant symptom and resembles mercurial tremor. It is, however, a wilder movement. The speech defects are different in the two conditions. In disseminated sclerosis the speech is syllabic, while in mercurial poisoning it is more of a stuttering defect.

In paralysis agitans the tremor is much more marked on movement, sometimes occurs only then; there is rigidity of the limbs and stolid countenance with paroxysmal flushing, symptoms usually sufficiently marked to render the diagnosis a matter of but little difficulty. When the chief symptom of lead-poisoning is tremor, it may be difficult to differentiate it from mercurial poisoning. The other symptoms of lead-poisoning are usually sufficiently marked to enable a conclusion to be formed, the lead line, the colic, the localization of the paresis being the more important.

In cases of doubt, an examination of the urine for mercury or other metals should be made.

PROGNOSIS.

In the great majority of cases of chronic mercurial poisoning recovery takes place, provided the cause is removed.

It is only in extreme cases of mercurial encephalopathia that a fatal ending may be the direct result of the poisoning. Such a state is very rare and is the outcome of the continuous absorption of the metal extending over many months or years.

The tremor may not completely pass away in chronic cases, but this does not indicate any danger to life.

TREATMENT.

It is in the first place essential that the patient should give up his occupation if it is one in which he is compelled to employ mercury, in any form.

For the promotion of the elimination of this metal we have to resort again to the employment of the iodide of potassium. It appears to be even more efficacious in mercurial than in either arsenical or lead poisoning. It is probable that mercury is not bound to the tissues in such an intimate manner as lead or arsenic. It is therefore advisable to begin with small doses, three to five grains three times daily. In this way there will be less danger of throwing quantities of the metal that may do acute injury into the circulation.

The bowels should be regularly moved by aloes or cascara. It will also be advisable to give an occasional saline to insure complete intestinal evacuation. The employment of diuretics to promote renal elimination may be a judicious practice.

For the anæmia iron should be used, and for the emaciation, in addition to good food and fresh air, cod-liver oil is strongly indicated.

It is a question whether we have any medicinal agents which have any influence in lessening the tremor attending mercurial poisoning or any other disease. I have seen some beneficial action following the administration of veratrine in cases of paralysis agitans; doses of $\frac{1}{100}$ grain given once or twice daily have had power apparently in lessening the tremors. The same dose would be appropriate for mercurial tremor.

SILVER.

Chronic silver-poisoning, argyria, may be due to the injudicious employment of the metal as a medicinal agent or to its use in certain trades.

Poisoning may result from either the internal or the local application of the metal, the former being much more common than the latter. Now that silver nitrate is rarely employed in epilepsy or chronic degenerative diseases of the spinal cord, argyria is a very rare affection. We have met with only two instances, both the result of silver used for lengthened periods in the treatment of epilepsy. It had been taken in both instances by the patients themselves without the advice of a medical man.

Gowers states that he has met with four cases of argyria: in two the silver was used for the treatment of epilepsy; in one it was used for syphilitic tumor of the spinal cord; in the fourth, the silver was taken as a "dinner pill."

Argyria as the result of silver used in the arts is very rare. Dr. W. F. Hamilton, of Montreal, reports two cases that he had an opportunity of seeing in Professor Jaksch's clinic. The patients were both middle-aged men, who had been occupied for twenty years in the manufacture of Bohemian glassware. Different silver solutions were used for staining the glass. In the process they drew the silver solutions into long tubes held in their mouths. There was intense pigmentation of the visible surfaces, skin and mucous membranes alike. As one observed the face and head they seemed of an intense blue-gray color, which deepened over the nose and forehead, and involved the exposed mucous membrane of the mouth and eyes, so that scarcely any difference of color was discernible between these parts and the skin. The surface shone as the light fell upon it, and the hair, once of a deep brown, seemed to share in the general glancing bluish-gray pigmentation. The mouth, the tongue, gums, uvula, and epiglottis were almost as deeply pigmented as the skin. The teeth were black. The pigmentation gradually diminished toward the extremities. Nothing further was remarkable about these cases, with the exception that one was a subject of arterio-sclerosis, but this was not referable to the action of the silver.

The discoloration of the skin, which is the characteristic sign of argyria, is due to a deposit of silver beneath the Malpighian layer. The darkening of the teeth by silver is said to occur before that of other parts.

In addition to the pigmentation of the skin and mucous membranes silver induces but very rarely paralysis and parenchymatous and interstitial changes in the liver and kidneys; little, however, is known about these changes.

Gowers reports a very interesting case of paralysis closely resembling lead paralysis as the result of silver. The patient was a man forty-four years of age, who had been taking a dinner pill composed of a silver salt more or less regularly for twelve years. He estimated that he had taken about six pills a month during the twelve years. About the beginning of the twelfth year, a dusky tint of the face attracted attention, but its nature was not suspected until it had deepened. Shortly afterward the extensors of the right hand became paralyzed, soon followed by the same changes in the extensors of the left hand. The affected muscles were atrophied. The reaction of degeneration was present. There was evidence of chronic Bright's disease. The man had had also several attacks of gout. Under treatment the patient improved slightly, but died a few months later from cancer of the liver.

The prognosis in cases of argyria is good as far as life is concerned, but once pigmentation is established it is permanent.

Parenchymatous changes in the internal organs will influence the ultimate prognosis.

The only treatment is a preventive one. As the pigmentation depends on the amount of silver taken, and not on the time occupied in taking it, it is all-important that silver should never be given for any lengthened period. Gowers considers that pigmentation begins when about an ounce of a salt of silver has been taken during any period of time.

PHOSPHORUS.

Acute Poisoning.

It is important to consider acute phosphorus-poisoning, owing to the close resemblance of the symptoms to certain diseases.

I am indebted almost entirely for the substance of this article to the admirable description of phosphorus-poisoning by Professor v. Jaksch in Nothnagel's "Specielle Pathologie und Therapie."

The frequency of acute phosphorus-poisoning in Germany and Austria is due to the prevalent use of this agent for suicidal purposes. Matches made with yellow phosphorus are in common use, and the ends are used by many for the purpose of committing suicide. Each stick contains about 0.0005 gm. ($\frac{1}{130}$ grain) and the phosphorus from one hundred sticks is a fatal dose.

It has been noted that there has been a marked increase during the past few years in the number of cases of phosphorus-poisoning. In v. Jaksch's clinic in Prague during the four years ending in December, 1893, no less than 28 cases of this form of poisoning came under observation.

Phosphorus-poisoning is rare in England and America.

SYMPTOMS.

Nausea, vomiting, and abdominal pain are among the first symptoms of acute phosphorus-poisoning. They may set in very quickly or there may be a delay of some hours after the ingestion of the mineral. They pass away in the course of two or three days.

Icterus, appearing during the course of a few days, is a characteristic symptom of phosphorus-poisoning. Its intensity is usually an index of the severity of the poisoning.

Simultaneously with the appearance of icterus, an enlargement of the liver in all directions can be detected by palpation and percussion. Professor v. Jaksch found the alkalinity of the blood diminished, and also a temporary increase in volume of the red blood corpuscles, a condition upon which he lays particular stress.

The changes found in the urine are also of great importance. It contains even in a few hours after the ingestion of the poison a small quantity of albumin. When icterus appears bile pigment is met with. Hyaline, fatty, and blood-casts are present. Leucin and tyrosin have been very rarely found.

Peptonuria is an occasional occurrence in phosphorus-poisoning. Münzer's observations in v. Jaksch's clinic show that the total nitrogenous elimination by the kidneys is diminished during the first few days, and afterward it is greatly increased in quantity. The elimination of ammonia is largely increased according to the same authority. He also found abnormal acids in the urine.

Certain nervous symptoms of a grave character may supervene during the first few days of phosphorus-poisoning, such as somnolence, coma, delirium, and general convulsions. Death may occur in a comatose state or suddenly from heart failure.

PATHOLOGICAL ANATOMY AND PATHOLOGY.

In the cases that run a rapidly fatal course, the odor of phosphorus can be detected in all the organs. Large and small hemorrhages are met with in the skin, serous and mucous membranes, muscles, etc. The mucous membrane presents traces of inflammation. The tissues are bile-stained.

The liver is greatly increased in size. Jaksch points out that if death occurs very early the liver may be found normal in size, or even atrophied. The spleen in his cases was almost invariably normal in size. He contends that splenic enlargement is not a feature of phosphorus-poisoning as is commonly supposed.

The heart, kidneys, muscles, glands of the stomach, and blood-vessels are found in a state of advanced fatty degeneration. The fatty degeneration is due to a rapid destruction of the albuminous tissues as well as to inefficient oxidation. There is a deficiency in the absorption of oxygen and the elimination of carbonic acid, together with a great increase in the elimination of urea.

The profound changes brought about in the organism are therefore due to destructive metabolic processes. The exact manner in which they are induced is, however, unknown.

DIAGNOSIS.

Acute or subacute phosphorus-poisoning presents certain features resembling acute yellow atrophy. The latter is characterized by the almost invariable presence of severe nervous symptoms, as delirium, convulsions, coma, etc.; although met with they are very uncommon in phosphorus-poisoning. In the latter the liver increases in size, while in the former it steadily diminishes. Jaksch reports a case where during life, and even after death on autopsy, it was impossible to make a diagnosis. Dr. Vivian Poore goes so far as to say that the great majority of cases of acute yellow atrophy of the liver are instances of phosphorus-poisoning and not of an independent disease.

In cases of very acute hypertrophic cirrhosis of the liver the symptoms closely resemble those of subacute phosphorus poisoning.

Jaksch considers that the absence of enlargement of the spleen in phosphorus-poisoning and its presence in hypertrophic cirrhosis of the liver will lead to a correct conclusion.

Certain forms of septic infection may present a resemblance to phosphorus-poisoning. Rigors, septic pyrexia, and enlargement of the spleen attend the former, but are not met with in the latter.

In all cases of doubt the vomited matters and fæces should be examined for phosphorus, for the details of which the reader is referred to works on chemistry.

PROGNOSIS.

The smallest fatal dose of phosphorus in v. Jaksch's series of cases was about 0.25 gm., while recovery followed the very large dose of 0.75 gm. In 39 cases the mortality was 38 per cent. Death may take place suddenly, many days after the subsidence of the acute gastro-intestinal symptoms, from heart failure (fatty degeneration). As unfavorable symptoms may be mentioned the early appearance of icterus; the presence of delirium or coma; the rapid increase in size of the liver; and hemorrhages from the mucous membranes or into the skin.

TREATMENT.

In acute cases the stomach should be freely washed out by means of the stomach-pump. If this is not at hand, then a brisk emetic of copper sulphate should be given. The washings should be repeated until the contents have lost all odor of phosphorus.

It is all important to avoid the administration of fats and oils and all substances containing them, as they dissolve any phosphorus and therefore assist in its absorption.

The internal administration of copper sulphate is also to be recommended as a chemical antidote; it forms the insoluble phosphate of copper. A few minims of a one-per-cent. solution can be frequently given.

Jaksch recommends the continuous administration, after the acute symptoms have subsided, of large doses of sodium carbonate to antagonize the acid intoxication which is considered by some to be at the bottom of the destructive metabolic changes induced by the phosphorus.

The intestines should be cleaned out by the internal administration of salines and copious rectal washings.

The hypodermic injection of camphor oil is suitable to meet the threatened cardiac failure. It should not, of course, be administered by the stomach.

The diet should be a mixed one, and the patient should be urged to take as much as possible, in order to make up for the destructive process going on in the albuminous tissues. To attain the same end the inhalation of oxygen would be also advisable.

Chronic Poisoning.

The most important pathological change met with as the result of the slow and continuous action of phosphorus is necrosis of the jaw due to a periostitis. This condition is met with only in match-makers, and owing to improved sanitary measures, it is now a very uncommon affection. The lower is more frequently affected than the upper jaw. The periostitis generally spreads from carious teeth.

The *treatment* is purely surgical.

Jaksch mentions that the long-continued internal use of phosphorus as a medicine may give rise to chronic parenchymatous and interstitial changes in the liver and kidneys. Nothing definite concerning this possible result of the medicinal use of phosphorus is known. It is, however, well to bear it in mind, when treating cases of rickets, etc., with phosphorus.

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INDEX TO VOLUME III.

Acids, poisoning by, 568
Aconine, 501
Aconite, poisoning by, 499
    fatal dose, 503
    illustrative cases, 504
    post-mortem appearances, 507
    symptoms, 502
    treatment, 506
Aconitine, 501
Æthusa cynapium, 539
Ague, brass-founders', 377
Air, effects of rarefied, 202, 217, 449
Albuminuria, acute alcoholic, 11
Alcohol, influence of, in the etiology of telegrapher's cramp, 476
Alcoholism, 6
    acute, 7; mania à potu, 11; delirium tremens, 13; epilepsy, 21; paralysis, 23; chronic, 33; pathology and post-mortem appearances, 39; treatment, 51; ginger, eau-de-Cologne, capsicum, and lavender inebriety, 67
Alcoholism, acute, 7
    coma, 7
    delirium tremens, 13
    epilepsy, 21
    gastric catarrh in, 10
    insanity of, 11
    mania à potu, 11
    melancholia in, 13
    pathology of, 39
    paralysis, 23
    pneumonia in, 11
    post mortem appearances of, 39
    treatment, 33
Alcoholism and Drug Habits, 3
    definition of terms, 4; toxic effect of articles taken to excess, 6; alcoholism, 6; acute alcoholism, 7; chronic alcoholism, 33; pathology of drunken acts, 44; ginger, eau-de-Cologne capsicum, and lavender inebriety, 67; morphinism, 70; chlorodynism, 80; cannabism, 88; chloralism, 89; chloroformism, 92; etherism, 95; cocainism, 99; gelsemiumism, 100; sulphonalism, 101; paraldehyde habit, 101; antipyrin habit, 103; arsenicism, 104; geophagy, 106; tobaccoism, 106; theinism, 112; caffeinism, 114; narcomania, 115; relations of inebriety to insanity, 116; periodical inebriety, 119; habitual or constant inebriety, 122; social and solitary inebriety, 123; etiology of inebriety, 123; pathology, 130; treatment, 131; medico-legal relations, 131; relation of inebriety to insurance, 136; bibliography, 137
Alcoholism and opium addiction contrasted, 72
    and tobaccoism, relations of, 111
    bibliography, 137
    capsicum inebriety, 69
    cardialgia in, 74
    chronic, 33
        post-mortem appearances of, 40
        symptoms, 34
    definition of, 5, 6
    eau-de-Cologne inebriety, 69
    ginger inebriety, 67
    lavender inebriety, 70
    modus operandi of injury to the brain in, 47
    pathology, 39
        of the tissue-crave, 50
    perversion of the moral sense in, 37, 47
    treatment, 51
        baths, 62

Alcoholism, treatment, cardiac remedies in, 54
    climatic, 60
    dietetic, 52, 63
    electricity, 62, 65
    gold, 65
    hyoscyamus, 54
    hypnotism, 56
    hypodermic injections, 53
    institutions, 59
    massage, 62, 65
    moral influence of abstention in the physician, 60
    nux vomica, 64
    occupation, 65
    of the predisposition, 62
    phenacetin, 54
    quinine, 64
    "specifics," 55
    strychnine, 54, 64
    suggestion, 57
    tobacco, 65
    tonics, 52, 63
    total withdrawal, 51
    trional, 54
Alcoholomania, definition of, 5
Alkalies, poisoning by, 582
    industrial, 404
Alphabet, the Morse, 474
Amalgam method of plating, dangers of, 349
Amblyopia, tobacco, 108
Ammonia, poisoning by, 583
    illustrative cases, 584
    symptoms, 584
    treatment, 585
Anæsthesia, influence of, upon the induction of shock, 147
Anamirta panniculata, 520
Aniline, poisoning by, industrial, 397
    symptoms, 399
    treatment, 400
Antipyrin, poisoning by, 103
Anthracosis, 419
Anthrax, 428
    external form, 428
    internal form, 429
    prevention, 430
    symptoms, 428
    treatment, 431
Aphonia from laryngeal fatigue, 459
Apoplexy, pulmonary, 260
    solar, 258, see *Heat-stroke*
Argyria, 616
Arsenic, poisoning by, 104, 606
    acute, 104, 606
    chronic, 104, 607
    elimination, 608
    etiology, 606, 607
    industrial, 342
    pathological anatomy and pathology, 606, 609
    symptoms, 345, 606, 608
    treatment, 104, 347, 607, 610
Arsenic-eating, 105
Arsenicism, 104, 606
Arterio-sclerosis in lead-poisoning, 597, 601
Arthritis deformans, diagnosis of, from osteomalacia, 248
Artificial flower makers, diseases of, 344, 358, 482
Asphyxia congelatorum, 289, see *Frost-bite*
Asphyxia, local, from cold, 299
    solar, 258, see *Heat-stroke*
Asthma, grinder's, 415
    potter's, 423
Atmosphere, effects of diminished pressure of the, 202, 217, 449
Atropa belladonna, 507
Atrophy, professional muscular, 483
    progressive muscular, diagnosis of, from acute alcoholic paralysis, 29
Atropine, poisoning by, 507, see *Belladonna*

BALLOON ASCENTS, effects of, 221
Belladonna, poisoning by, 507
    fatal dose, 511
    illustrative cases, 511
    symptoms, 509
    treatment, 512
Bhang, addiction to, 88
Bittersweet, 553
Bleaching powder, risks of the makers of, 384
Boiler-makers, deafness of, 451
Bones, cancerous infiltration of the, diagnosis of, from osteomalacia, 248
    softening of the, in osteomalacia, 238
Brain, alcoholic lesions in the, 41

Brain, concussion of the, diagnosis of, from shock, 162
Brass, poisoning by, 375
Brass-founder's ague, 377
Bricklayer's spasm, 483
Brick-makers, diseases of, 387, 391
Bromine, poisoning by, industrial, 385
Bronze, poisoning by, 375
Bronzing by amalgam, dangers of, 350
Brucine, 558

CAFFEINISM, 114
  treatment, 115
Caisson disease, 437
    causes, 440
    pathology, 443
    prevention, 446
    symptoms, 441
    treatment, 447
Calabar bean, poisoning by, 514
    symptoms, 516
    treatment, 517
Cancer of the bones, diagnosis of, from osteomalacia, 248
Cannabism, 88
Caoutchouc, vulcanization of, injurious effects of the process of, 392
Capsicum addiction, 69
Carbolic acid, poisoning by, 573
    fatal dose, 575
    illustrative cases, 575
    symptoms, 574
    treatment, 575
Carbon bisulphide, poisoning by, diagnosis, 394
    industrial, 391
    prevention, 396
    symptoms, 393
    treatment, 396
Carbon dioxide, poisoning by, industrial, 390
    treatment, 391
Carbon monoxide, poisoning by, industrial, 389
    treatment, 390
Cardialgia of opium users, 74
Catarrh, acute alcoholic gastric, 10
Chaps from cold, 299
Chilblain, 299
  resemblance of, to Raynaud's disease, 300
Chilblain, treatment, 305
Child-labor, evils of, 322
  laws regulating, in England, 323
    in France, 327
    in the United States, 328
Chloral hydrate, poisoning by, 89, 585
    acute, 89
    chronic, 90
    fatal dose, 586
    industrial, 384
    symptoms, 586
    treatment, 90, 92, 586
Chloralism, 89
Chlorodyne, addiction to, 80
  poisoning by, 80
Chloroform, addiction to, 94
  fall of temperature after protracted anæsthesia by, 148
  poisoning by, 92
    acute, 92
    chronic, 94
Chloroformism, 92
Christmas rose, 535
Chrome-yellow, danger in the preparation of, 382
Chromium, poisoning by, industrial, 382
    symptoms, 383
Cicuta maculata, 539
  virosa, 539
Cicutine, 539
Cicutoxin, poisoning by, 539
Cinnabar mining, dangers of, 348
Clay dust, injurious effects of, 421
Clay-eating, 106
Climate, relation of, to inebriety, 121, 126, 130
Coal dust, injurious effects of, 419
Coca, poisoning by, 99, 517
Cocaine, addiction to, 99
  poisoning by, 99, 517
    acute, 99
    chronic, 99
    fatal dose, 519
    illustrative cases, 519
    pathology, 99
    symptoms, 99, 518
    treatment, 520
Cocainism, 99
Cocculus indicus, poisoning by, 520
    symptoms, 522
    treatment, 522

Codeine, poisoning by, 552
Coffee, poisoning by, 114
Colchicine, 524
Colchicum autumnale, 523
    poisoning by, 523
        illustrative cases, 525
        symptoms, 525
        treatment, 526
Cold, effects of, 289, 436, see *Frost-bite*
Cold-stroke, 290
Colic, lead, 362, 596
Colica pictonum, 363
Collapse, distinction of, from shock, 144, 155
Cologne water inebriety, 69
Coma, acute alcoholic, 7
Compositors, diseases of, 357, 483
Compressed air, injurious effects of, 437
Concussion of the air, injurious effects of, 451
Congelatio, 289, see *Frost-bite*
Coniine, poisoning by, 536, see *Hemlock*
Conium maculatum, 536
Convalescence, advantages of a sea voyage during, 196
Convulsions in lead-poisoning, 599
Copper, poisoning by, 611
        acute, 611
        chronic, 612
        industrial, 373
        symptoms, 377, 611, 612
        treatment, 378, 611, 612
Coryza, chromium, 383
Cotton dust, effects of, 425
COUNCILMAN, W. T., on osteomalacia, 233
Cramp, telegrapher's, writer's, etc., see *Telegrapher's cramp*, etc.
Crime, drunkenness as an excuse for, 13
Curare, poisoning by, 526
        symptoms, 527
        treatment, 527
Cutlers, hephæstic hemiplegia in, 481
Cyanide of potassium, poisoning by, 577, see *Hydrocyanic acid*

DATURA stramonium, 555
Daturine, 556
Death from fright, 152
Deformities resulting from occupation, 494
        osteomalacia, 236
Degeneration-reaction in lead-poisoning, 598
Delirium in lead-poisoning, 365, 599
Delirium tremens, 13
    absinthal, 16
    diagnosis of, from mania à potu, 12
    diagnosis of, from the delirium of chronic alcoholism, 16
    diagnosis of, from the delirium of acute diseases, 16
    forms of, 14
    non-alcoholic imitative, 15
    prognosis, 15
    symptoms, 13
    treatment, 16
Dementia, alcoholic, 38
        diagnosis of, from general paralysis, 38
Digitalein, 529
Digitalin, 529
Digitalis, poisoning by, 528
        fatal dose, 531
        illustrative cases, 531
        symptoms, 530
        treatment, 532
Digitonin, 529
Digitoxin, 529
Dirt-eaters, 106
Dressmakers, diseases of, 357, 483
Drug habits, 3
    alcohol, 6
    antipyrin, 103
    arsenic, 104
    bibliography, 137
    caffeine, 114
    cannabis indica, 88
    capsicum, 69
    castor oil, 106
    chloral, 89
    chlorodyne, 80
    chloroform, 92
    clay, 106
    cocaine, 99
    cod-liver oil, 106
    cologne water, 69
    Epsom salts, 106
    ether, 95
    gelsemium, 100
    ginger, 67
    iodine, 106
    lavender, 70

Drug habits, morphine, 70
  opium, 70
  paraldehyde, 101
  paraffin, 106
  potassium bicarbonate, 106
  potassium iodide, 106
  sulphonal, 101
  theine, 112
  tobacco, 106
Drunken acts, pathology of, 44
Drunkenness, definition of, 5, see *Inebriety*
Dupuytren's contraction due to occupation, 495
Dust, injurious effects of, upon workmen, 411
  metallic, effects of, 412
  mineral, effects of, 418
  organic, effects of, 424
Dyers, diseases of, 343, 382, 398
Dysgraphia, 462
Dyspnœa, heat, 253, see *Heat-stroke*
  in mountain sickness, 217

EARTH-EATING, 106
Electricity, accidents from, 408
  execution by, 151, 409
  fatal action of, on the human body, 409
  shock caused by, 151
Encephalopathy, lead, 599
Engraver's cramp, 482
Epilepsy, acute alcoholic, 21
Epsom salts, addiction to, 106
Erethismus tropicus, 253, see *Heat-stroke*
Eserine, poisoning by, 514, see *Calabar bean*
Erythroxylon coca, 517
Ether, fall of temperature after continued administration of, 148
  poisoning by, 95
  test by, prior to operation upon a person suffering from shock, 148
Ether-drinking, history of, 96
Etherism, 95
  pathology, 98
  symptoms, 98
  treatment, 98
Exhaustion, heat, 258, see *Heat-stroke*
Expiration, effects of rarefied air upon, 217

FACTORY ACTS IN ENGLAND, 323
Fat embolism, diagnosis of, from shock, 163
Febris thermica, 258, see *Heat-stroke*
Fecundity, influence of cigar-making upon, 432
Felt-hat makers, mercurial poisoning in, 350
Fever, ardent, 253, see *Heat-stroke*
  shoddy, 427
  sun, 253, see *Heat-stroke*
  thermic, 258, see *Heat-stroke*
File-cutters, diseases of, 360, 413
Flax dust, effects of, 425
Fork-grinding, injurious effects of, 415
Foxglove, 528
Freezing, 289, see *Frost-bite*
Fright, shock from, 151
Frigore, ambustio ex, 289, see *Frost-bite*
Frost-Bite, 289
  definition, 289; etiology, 291; symptoms, 294; morbid anatomy, 297; chilblain, 299; treatment, 300; sequelæ, 306; bibliography, 306
  bibliography, 306
  chilblain, 299
  etiology, 291
  industrial, 436
  morbid anatomy, 297
  resemblance of, to Raynaud's disease, 300
  sequelæ, 306
  symptoms, 294
  treatment, 300
    of chilblain, 305
    of gangrene from, 304
    of secondary inflammation, 302
    of ulceration following, 304
  typical cases of, 294

GALENA, lead-poisoning among miners of, 356
Galvanized iron, danger to makers of, 380
Gangrene from freezing, treatment of, 304
Gas, illuminating, see *Illuminating gas*
Gastritis, acute alcoholic, 10
Gelsemine, 533
Gelsemium, addiction to, 101
  poisoning by, 100, 533

Gelsemium, poisoning by, acute, 100
        chronic, 101
        illustrative case, 534
        symptoms, 100, 534
        treatment, 100, 534
Gelsemiumism, 101
Geophagy, 106
GIHON, ALBERT L., on frost-bite, 287
    on heat-stroke, 251
    on seasickness, 173
Gilding by amalgam, dangers of, 349
Ginger-drinking, 67
Glass-cutters, diseases of, 358
Gout, lead-poisoning in relation to, 365, 597
Graphospasmus, 462
Grinder's disease, 413
    post-mortem appearances, 417
    prevention, 417
    symptoms, 415
    treatment, 418
Grinders, fatigue palsy in, 481
Gums, blue line on the, from lead, 596
Gunja, addiction to, 88

HABITS, drug, 3, see *Drug habits*
Hair, danger of anthrax infection to workers in, 428
    discoloration of, by copper, 377
Hammer palsy, 481
Hashish, addiction to, 88
Heart, fireman's, 255, 283
    irritability of, following heat-stroke, 255, 283
Heat, effects of excessive, 253, see *Heat-stroke*
Heat-stroke, 253
    definitions, 254; symptoms, 258; causation, 272; morbid anatomy, 275; prophylaxis, 276; treatment, 277; sequelæ, 283; bibliography, 285
    and sunstroke, distinguished by some writers, 261
    bibliography, 285
    cardiac form, 260
    causation, 272
    cerebro-spinal form, 259
    definition, 254
    forms of, 258, 259
    heart trouble following, 255, 283
Heat-stroke, hyperpyrexia in, 259
    in children, 271
    industrial, 433
    insanity following, 284
    local, 272
    meningeal form, 259
    morbid anatomy, 275
    odor of, 259, 268
    pathology, 272
    prophylaxis, 276
    pulmonary apoplexy, 260
    sequelæ, 283
    statistics of, in the U. S. Army and Navy, 255
    symptoms, 258, 435
    treatment, 277, 436
    typical cases of, 263
    visual disturbances following, 284
Hellebore, poisoning by, 535
        symptoms, 535
        treatment, 536
Helleborein, 535
Helleborin, 535
Hemiplegia, cerebral, diagnosis of, from acute alcoholic paralysis, 31
    hephæstic, 481
Hemlock, poisoning by, 536
        fatal dose, 538
        illustrative case, 538
        symptoms, 537
        treatment, 538
Hemorrhage, effect of, upon the production of shock, 150
    tendency to, at high altitudes, 207
Hemp, Indian, chronic poisoning by, 88
Henbane, poisoning by, 539, see *Hyoscyamus*
Heredity of inebriety, 128
Hides, danger of anthrax infection to handlers of, 429
Horse-nettle, 553
Housemaid's knee, 496
Hydrargyrism, 613, see *Poisoning by mercury*
Hydrochloric acid, poisoning by, 571
        illustrative cases, 572
        symptoms, 572
        treatment, 572
Hydrocyanic acid, poisoning by, 576
        illustrative cases, 579

Hydrocyanic acid, poisoning by, industrial, 386
    symptoms, 578
    treatment, 579
Hyoscine, 540
Hyoscyamine, 540
Hyoscyamus, poisoning by, 539
    illustrative cases, 541
    symptoms, 541
    treatment, 542
Hysteria, toxic, 394
  toxic, in alcoholism, 30

ICTUS CALORIS, 253, see *Heat-stroke*
  solaris, 253, see *Heat-stroke*
Igasurine, 558
Illuminating gas, poisoning by, 587
    illustrative case, 589
    industrial, 396
    symptoms, 588
    treatment, 590
Impetigo glycosique, 435
India-rubber workers, injury to, from bisulphide of carbon, 391
Inebriety, 115
  alcohol, 6
  bibliography, 137
  capsicum, 69
  civil law aspects of, 135
  commercial periodic, 121
  constant, 122
  criminal jurisprudence of, 131
  definition of, 5
  eau-de-Cologne, 69
  etiology, 123
    age, 124
    associated indulgence, 129
    chest diseases, 129
    climate, 126
    diet, 129
    education, 127
    epilepsy, 129
    heredity, 128
    idleness, 130
    intoxicants, 130
    marriage, 128
    menopause, 130
    menstrual disorders, 130
    nerve shock, 130
    occupation, 128
    overwork, 130
Inebriety, etiology, pecuniary circumstances, 127
    puberty, 130
    race, 126
    religion, 125
    rheumatism, 129
    sex, 124
    syphilis, 129
    temperament, 129
    traumatism, 130
    worry, 130
  forms of, 119
  ginger, 67
  habitual, 122
  lavender, 70
  legal responsibility in, 131
  medico-legal relations of, 131
  menstrual, 119
  morphine, 70
  opium, 70
  pathology, 130
  periodical, 119
    premonitory symptoms of the paroxysm, 122
  predisposing causes, 124
  relation of, to insanity, 116
    to insurance, 136
  seasonal periodic, 121
  social, 123
  solitary, 123
  testamentary capacity affected by, 135
  treatment, 131
Infants, cold-stroke in, 290
  heat-stroke in, 271
Insanity, acute alcoholic, 11
  due to hashish addiction, 88
  following heat-stroke, 284
  relations of, to inebriety, 116
Insolatio, 253, see *Heat-stroke*
Insolation, local, 272
Inspiration, effects of rarefied air upon, 218
Insurance, relation of inebriety to, 136
Iodine, addiction to, 106
  poisoning by, industrial, 385
Intestines, shock following injury to the, 150
Intoxication, alcoholic, 7
    pathology of, 44, see also *Inebriety*
Intoxication-mania, 5

JAMESTOWN WEED, 555
Jasmine, yellow, 100, 533, see *Gelsemium*
Jaundice, acute alcoholic, 11
Jaws, necrosis of the, from phosphorus, 371
Jessamine, yellow, 100, 533, see *Gelsemium*
Jimson weed, 555

KERR, NORMAN, on alcoholism and drug habits, 1
Kibes, 299, 305
Kidney, lesions of the, in lead-poisoning, 602
Kinetia, 175, see *Seasickness*

LABOR, child, evils of, 322
  laws regulating, in England, 323
    in France, 327
    in the United States, 328
Lace-makers, lead-poisoning among, 358
Larynx, concussion of the, 145
  fatigue neurosis of the, 458
Lavender drinking, 70
Lead, paralysis from, 363, 598
  poisoning by, 593
    diagnosis, 602
    elimination, 605
    industrial, 355
    local, 362
    mode of absorption, 595
    pathological anatomy and pathology, 601
    prevention, 366, 604
    prognosis, 603
    relation of gout to, 365, 597
    sources of, 593
    symptoms, 362, 595
    treatment, 367, 604
v. LIEBIG, GEORG, on mountain sickness, 199
Life insurance, relation of inebriety to, 136
Linemen, electrical, danger of fatal shocks to, 408
Lithographers, diseases of, 344,
LLOYD, JAMES HENDRIE, on diseases of occupations, 309
Locomotive engineers and firemen, effects of occupation upon, 496
Locomotor ataxia, diagnosis of, from acute alcoholic paralysis, 28
Locomotor ataxia, lead-poisoning resembling, 364
Lungs, condition of the, in mountain sickness, 219
  disease of the, among cotton workers, 425
    among flax workers, 426
    among grinders, 415
    among miners, 418
    among potters, 423
  increased elastic tension of the, in mountain sickness, 217, 225, 227

MAGNESIUM sulphate, addiction to, 106
Malignant pustule, 428
Mania à potû, 11
Mania, intoxication, 5
Match-makers, diseases of, 368, 387
Melancholia, acute alcoholic, 13
Meningitis, spinal, diagnosis of, from acute alcoholic paralysis, 30
Menispermum cocculus, 520
Menopause, impulse to inebriety at the, 130
Menstruation, narcomania of, 119
Mercury, poisoning by, 613
    diagnosis, 615
    industrial, 348
    pathological anatomy and pathology, 614
    prognosis, 616
    sources, 613
    symptoms, 352, 613
    treatment, 354, 616
Methyl-coniine, 537
Milker's spasm, 483
Miners, diseases of, 342, 348, 356, 379, 381, 412, 418, 434, 449, 484, 495
Miner's nystagmus, 484
  phthisis, 418
Mirror-makers, mercurial poisoning in, 351
Mogigraphia, 462
Monkshood, 499
Morphine, mode of administration as affecting rapidity of poisoning by, 77
  poisoning by, 71, see *Opium*
Morphinism, 70, see *Opium, addiction to*
Morphinomania, definition of, 5
Morse alphabet, 474
Mountain fever, 449

- **Mountain Sickness,** 201
  - symptoms, 202; history, 210; an attempt to explain the symptoms, 215; summary, 225; acclimation, 226; diagnosis, 227; prognosis, 228; treatment, 228; bibliography, 229
- Mountain sickness, acclimation, 226
  - altitude of occurrence of, 202
  - attempt to explain the symptoms, 215
  - bibliography, 229
  - cause, 215, 225
    - resides in individual only, 213
  - circulation in, 223
  - condition of the lungs in, 219
  - diagnosis, 227
  - dyspnœa, 217, 223
  - expiration in, 217
  - history, 210
  - immunity of natives of elevated regions, 226
  - increased elastic tension of the lungs in, 217, 225, 227
  - inspiration in, 218
  - nature, 215
  - prognosis, 228
  - pulse in, 223
  - respiration in, 217
  - summary, 225
  - symptoms, 202
    - attempt to explain the, 215
    - increased by fatigue and hunger, 209, 221
  - theory of, 215
  - treatment, 228
- Mules (chilblains), 299, 305
- Musicians, diseases of, 459, 482
- Myeloma, diagnosis of, from osteomalacia, 248

- NAPELLINE, 501
- Narcomania, 115, see *Inebriety*
  - definition of, 5
- Narcotine, 544
- Naupathia, 175, see *Seasickness*
- Nausea of mountain sickness, 203
  - of seasickness, 176
- Necrosis, phosphorus, 371
- Needle-grinding, injurious effects of, 414
- Nephritis, acute alcoholic, 11
- Neurasthenia, alcoholic, 32
    - treatment, 54
  - from over-work, 488
  - in brain-workers, 490
  - symptoms, 490
  - treatment, 492
- Neuritis, arsenical, 346
  - diagnosis of certain forms of, from acute alcoholic paralysis, 29
  - progressive alcoholic, post-mortem appearances of, 43
- Neuroses, fatigue, 457
  - professional, 457
- Nightshade, 553
  - deadly, 507
- Nitric acid, poisoning by, 570
    - symptoms, 571
    - treatment, 571
- Nitro-benzene, poisoning by, industrial, 400
    - symptoms, 401
    - treatment, 402
- Nose, perforation of septum, in workers in chromium, 383
- Nux vomica, poisoning by, 557, see *Strychnine*
- Nystagmus, miner's, 484

- **Occupations, Diseases of,** 311
  - historical note, 311; conditions of occupation with reference to the prevalence of disease, 317; legal restraints upon the employment of women and children, 323; classification, 339; effects of injurious substances, 342; effects of injurious environment, 433; effects of injurious exercise, 457
- Occupations, diseases of, age factor in, 322
  - artificial-flower makers, 344, 358, 482
  - boiler-makers, 451
  - brass-workers, 359, 374
  - bricklayers, 483
  - brick-makers, 384, 387
  - bronzers, 350, 357, 374
  - caisson disease, 437
  - calico-printers, 361, 493
  - carpenters, 494
  - chromium workers, 382

Occupations, cigar-makers, 431
  classification of, 339
  clergymen, 459
  compositors, 357, 483
  conditions of occupation with reference to the prevalence of disease, 317
  copper-smiths, 374
  cutlers, 481
  deformities resulting from constrained attitudes, 494
  dentists, 494
  domestic servants, 496
  dressmakers, 357, 483
  dyers, 343, 382, 398
  effects of alkalies, 404
    aniline, 397
    anthrax bacillus, 428
    arsenic, 342
    bad sanitation and ventilation, 452
    bisulphide of carbon, 391
    bromine, 385
    chlorine, 384
    chromium, 382
    cold, 436
    compressed air, 437
    concussion of the air, 451
    constrained attitudes, 493
    copper, 373
    cyanogen compounds, 386
    dust, 411
    dust, metallic, 412
    dust, mineral, 418
    dust, organic, 424
    electricity, 408
    heat, 433
    illuminating gas, 396
    injurious environment, 341, 433
    injurious exercise, 342
    injurious substances, 341, 342
    iodine, 385
    lead, 355
    mercury, 348
    nitro-benzene, 400
    oxides of carbon, 389
    phosphorus, 367
    quinine, 405
    rarefied air, 449
    sewing machine, 486
    sulphur and its oxides, 386
Occupations, effects of tea, 406
    tin, 381
    tobacco, 431
    turpentine, 402
    zinc, 379
  electricians, 408
  engravers, 482, 494
  fatigue neuroses, 457
  felt-hat makers, 350
  file-cutters, 360, 413
  floor-planers, 496
  furniture polishers, 357
  galvanizers of iron, 380
  gilders, 349, 357
  glass-cutters, 358
  glazing, 359
  grinders, 413, 481
  historical note, 311
  India-rubber workers, 391
  jewellers, 360, 494
  lace-makers, 358
  lithographers, 344
  locksmiths, 495
  locomotive engineers and firemen, 496
  match-makers, 368, 387
  milkers, 483
  miners, 342, 348, 356, 379, 381, 412, 418, 434, 449, 484, 495
  mirror-makers, 351
  mountain fever, 449
  musicians, 459, 482
  neurasthenia from over-work, 488
  painters, 357, 402
  paper-hangers, 343
  paper-makers, 404
  photographers, 386
  pianists, 482
  plumbers, 357
  potters, 421, 454
  professional men, 490
  quarry-men, 420
  race factor in, 339
  relations of country to, 339
  roburite makers, 401
  roofers, 496
  seamstresses, 357, 483
  sex factor in, 323
  sheet-iron workers, 451
  shoemakers, 483, 496
  shot-makers, 344, 357

- Occupations, singers, 459
  - smelters, 343, 348, 356, 374, 379, 381, 389
  - stokers, 434
  - stone-cutters, 420
  - sugar refiners, 435
  - tailors, 483
  - tea-tasters, 406
  - telegraphers, 473
  - tin-plate makers, 381
  - tobacco-workers, 360, 431
  - type-founders, 357
  - type-setters, 357, 483
  - wall-paper makers, 343
  - weavers, 360, 424, 425, 427, 453, 489
  - white-lead makers, 360
  - wire-workers, 495
  - wool-sorters, 428
  - writers, 462
- Odor of the body and excretions in heat-stroke, 259, 268
- Oil of bitter almonds, poisoning by, 577, see *Hydrocyanic acid*
- Opium, addiction to, 70
    - "abstinence phenomena," 83
    - and alcoholism contrasted, 72
    - cardialgia in, 74
    - causes of, 78
    - course and termination, 81
    - pathology of, 72
    - prevalence of, 80
    - treatment, 82
    - treatment, length of time necessary for, 86
    - treatment, results of, 88
  - mode of administration as affecting rapidity of poisoning by, 77
  - "moderate" indulgence in, in the East, 76
  - poisoning by, 70, 542
    - fatal dose, 547
    - illustrative cases, 548
    - symptoms, 546
    - treatment, 549
- Opiumism, 70, see *Opium, addiction to*
- **Osteomalacia**, 235
  - deformities, 236; pathological anatomy, 238; etiology, 240; pathology, 231; relation of osteomalacia to disease of the ovaries, 244; diagnosis, 246; treatment, 248
- Osteomalacia, definition, 235
  - deformities, 236
  - diagnosis, 246
    - from arthritis deformans, 248
    - from bone tumors, 248
    - from multiple myelomata, 248
    - from partial dislocation of the bones of the pelvis, 248
  - etiology, 240
  - in animals, 239
  - pathological anatomy, 238
  - pathology, 241
  - puerperal, osseous tenderness in, 247
  - senile form of, 239
  - theories of, 241
    - congestion of the bones, 242
    - infections, 242
    - inflammation of the bones, 242
    - lactic acid, 241
    - ovarian disease, 244
    - trophoneurosis of the bone, 245
  - treatment, 248
- Ovaries, relation of osteomalacia to diseases of the, 244
- Oxalic acid, poisoning by, 580
    - fatal dose, 581
    - illustrative cases, 581
    - symptoms, 581
    - treatment, 582

- Painters, diseases of, 357, 402
- Papaver somniferum, 542
- Papaverine, 543
- Paper-hangers, arsenical poisoning of, 343
- Paper-makers, effects of the alkalies upon, 404
- Paraldehyde, addiction to, 102
  - poisoning by, 101
- Paralysis, acute alcoholic, 23
    - diagnosis, 26
    - diagnosis of, from anterior poliomyelitis, 30
    - diagnosis of, from cerebral hemiplegia, 31
    - diagnosis of, from disseminated sclerosis, 29
    - diagnosis of, from inflammatory diseases of the spinal cord, 30
    - diagnosis of, from lead palsy, 27

Paralysis, acute alcoholic, diagnosis of, from locomotor ataxia, 28
    diagnosis of, from progressive muscular atrophy, 29
    post-mortem appearances, 43
    symptoms, 23
    treatment, 33
  general, diagnosis of, from alcoholic dementia, 38
  hammer, 481
  Landry's, diagnosis of, from acute alcoholic paralysis, 29
  lead, 363, 598
    diagnosis of, from alcoholic paralysis, 27, 603
  mercurial, 353
  peripheral, following frost-bite, 306
  toxic hysterical, 30, 394
Pelvis, partial dislocation of the bones of the, simulating osteomalacia, 248
Periodicity, inebriate, 119
Pernio, 299, 305
  treatment, 305
Phenol, poisoning by, 573, see *Carbolic acid*
Phosphorus, poisoning by, 618
    acute, 618
    chronic, 621
    diagnosis, 620
    industrial, 367
    pathological anatomy and pathology, 619
    prevention, 372
    symptoms, 370, 619, 621
    treatment, 621
Phrenitis æstiva, 253, see *Heat-stroke*
Phthisis, miner's, 418
Physicians, general immunity of, from neurasthenia, 490
  opium addiction among, 78
Physostigma venenosum, 514
Physostigmine, poisoning by, 514, see *Calabar bean*
Pianist's cramp, 482
Picrotoxin, poisoning by, 520, see *Cocculus indicus*
Plumbism, see *Poisoning by lead*
Pneumonia, acute alcoholic, 11
Poisoning, 499
  aconite, 499; belladonna, 507, Calabar bean, 514; cocaine, 517; cocculus indicus, 520; colchicum, 523; curare, 526; digitalis, 528, gelsemium, 533; hellebore, 535; hemlock, 536; hyoscyamus, 539; opium, 542; solanum, 553; stramonium, 555; strychnine, 557; veratrum, 566; sulphuric acid, 568; nitric acid, 570; hydrochloric acid, 571; carbolic acid, 573; hydrocyanic acid, 576; oxalic acid, 580; potassa and soda, 582; ammonia, 583; chloral hydrate, 585; illuminating gas, 587; lead, 593; arsenic, 606; zinc, 611; copper, 611; mercury, 613; silver, 616; phosphorus, 618
Poisoning by acids, 568
  by aconite, 499
    illustrative cases, 504
    symptoms, 502
    treatment, 506
  by alkalies, 582
    industrial, 404
  by alcohol, 3
    acute, 7
    chronic, 33
  by ammonia, 583
    illustrative cases, 584
    symptoms, 584
    treatment, 585
  by aniline, industrial, 397
    symptoms, 399
    treatment, 400
  by antipyrin, 103
  by arsenic, 104, 606
    acute, 606
    chronic, 607
    elimination, 608
    etiology, 606, 607
    industrial, 342
    pathological anatomy and pathology, 606, 609
    symptoms, 345, 606, 608
    treatment, 347, 607, 610
  by atropine, 507, see *Belladonna*
  by belladonna, 507
    illustrative cases, 511
    symptoms, 509
    treatment, 512
  by brass, industrial, 375
  by bromine, industrial, 385
  by bronze, industrial, 375

- Poisoning by brucine, 558
  - by Calabar bean, 514
    - symptoms, 516
    - treatment, 517
  - by cannabis indica, 88
  - by carbolic acid, 573
    - fatal dose, 575
    - illustrative cases, 575
    - symptoms, 574
    - treatment, 575
  - by carbon bisulphide, 391
    - diagnosis, 394
    - prevention, 396
    - symptoms, 393
    - treatment, 396
  - by carbon dioxide, industrial, 390
    - treatment. 391
  - by carbon monoxide, industrial, 389
    - treatment, 390
  - by chloral hydrate, 89, 585
    - fatal dose, 586
    - symptoms, 586
    - treatment, 586
  - by chlorine, industrial, 384
  - by chloroform, 92
  - by chromium, industrial, 382
    - symptoms, 383
  - by cicutoxin, 539
  - by coca, 517
  - by cocaine, 99, 517
    - fatal dose, 519
    - illustrative cases, 519
    - symptoms, 518
    - treatment, 520
  - by cocculus indicus, 520
    - symptoms, 522
    - treatment, 522
  - by codeine, 552
  - by coffee, 114
  - by colchicum, 523
    - illustrative cases, 525
    - symptoms, 525
    - treatment, 526
  - by conium, 536, see *Hemlock*
  - by copper, 88, 611
    - acute, 611
    - chronic, 612
    - industrial, 373
    - symptoms, 377, 611, 612
    - treatment, 78, 611, 612
- Poisoning by curare, 526
    - symptoms, 527
    - treatment, 527
  - by cyanide of potassium, 577, see *Hydrocyanic acid*
  - by digitalis, 528
    - fatal dose, 531
    - illustrative cases, 531
    - symptoms, 530
    - treatment, 532
  - by eserine, 514, see *Calabar bean*
  - by ether, 95
  - by gelsemium, 100, 533
    - illustrative cases, 534
    - symptoms, 534
    - treatment, 534
  - by hashish, 88
  - by hellebore, 535
    - symptoms, 535
    - treatment, 536
  - by hemlock, 536
    - fatal dose, 538
    - illustrative case, 538
    - symptoms, 537
    - treatment, 538
  - by hydrochloric acid, 571
    - illustrative cases, 572
    - symptoms, 572
    - treatment, 572
  - by hydrocyanic acid, 576
    - industrial, 386
    - illustrative cases, 579
    - symptoms, 578
    - treatment, 579
  - by hyoscyamus, 539
    - illustrative cases, 541
    - symptoms, 541
    - treatment, 542
  - by illuminating gas, 587
    - illustrative cases, 589
    - industrial, 396
    - symptoms, 588
    - treatment, 590
  - by Indian hemp, 88
  - by iodine, industrial, 385
  - by lead, 593
    - elimination, 605
    - diagnosis, 602
    - industrial, 355
    - local, 362
    - mode of absorption, 595

Poisoning by lead, pathological anatomy and pathology, 601
 prevention, 366, 604
 prognosis, 603
 relation of gout to, 597
 sources of, 593
 symptoms, 362, 595
 treatment, 367, 604
 by mercury, 613
 diagnosis, 615
 industrial, 348
 pathological anatomy and pathology, 614
 prognosis, 616
 sources, 613
 symptoms, 352, 613
 treatment, 354, 616
 by morphine, 71, 545, see *Opium*
 by nitric acid, 570
 symptoms, 571
 treatment, 571
 by nitro-benzene, industrial, 400
 symptoms, 401
 treatment, 402
 by oil of bitter almonds, 577, see *Hydrocyanic acid*
 by opium, 70, 542
 fatal dose, 547
 illustrative cases, 548
 symptoms, 546
 treatment, 549
 by oxalic acid, 580
 fatal dose, 581
 illustrative cases, 581
 symptoms, 581
 treatment, 582
 by paraldehyde, 101
 by phenol, 573, see *Carbolic acid*
 by phosphorus, 618
 acute, 618
 chronic, 621
 diagnosis, 620
 industrial, 367
 pathological anatomy and pathology, 619
 prevention, 372
 prognosis, 620
 symptoms, 370, 619, 621
 treatment, 621
 by physostigmine, 514, see *Calabar bean*
Poisoning by picrotoxin, 520, see *Cocculus indicus*
 by potassa, 582
 by potassium cyanide, 576, see *Hydrocyanic acid*
 by prussic acid, 576, see *Hydrocyanic acid*
 by quinine, industrial, 405
 by silver, 616
 by soda, 582
 by solanine, 554
 by solanum, 553
 symptoms, 554
 treatment, 555
 by stramonium, 555
 symptoms, 556
 treatment, 557
 by strychnine, 557
 fatal dose, 563
 illustrative cases, 563
 symptoms, 561
 treatment, 564
 by sulphonal, 101
 by sulphur, industrial, 386
 by sulphuric acid, 568
 industrial, 388
 symptoms, 569
 treatment, 570
 by sulphurous acid, industrial, 387
 by tea, 112
 industrial, 406
 symptoms, 407
 by tobacco, 106
 industrial, 431
 by turpentine, industrial, 402
 symptoms, 402
 treatment, 403
 by veratrum, 566
 symptoms, 567
 treatment, 568
 by zinc, 611
 industrial, 379
 shock occurring in cases of, 155
Poliomyelitis, anterior, diagnosis of, from acute alcoholic paralysis, 30
Polyneuritis, diagnosis of, from acute alcoholic paralysis, 29
Poppy, 542
Potassa, poisoning by, 582
Potassium bicarbonate, addiction to, 106
Potassium cyanide, poisoning by, 576

Potters, diseases of, 421, 454
Professional men, neurasthenia in, 490
Professional neuroses, 457
Prostratio thermica, 253, see *Heat-stroke*
Prostration, 141
Prussic acid, poisoning by, 576, see *Hydrocyanic acid*
Pseudaconitine, 501
Pustule, malignant, 428

Quarrying, injurious effects of, 420
Quicksilver mining, dangers of, 348
Quinine, poisoning by, industrial, 405

Railway accidents producing shock, 148
Rarefied air, effects of, 202, 217, 449
Raynaud's disease, resemblance of, to chilblains, 300
Respiration, effects of rarefied air upon, 217
  in mountain sickness, 205
Rhinitis, chromium, 383
Rose, Christmas, 535

Salivation, mercurial, 352
Saturnism, see *Poisoning by lead*
Scrivener's palsy, 462
Seasickness, 175
  symptoms, 176; causation, 181; nature, 188; treatment, 189; therapeutic uses of a sea voyage, 195; bibliography, 196
Seasickness, bibliography, 196
  causation, 181
  fatal cases of, 181
  influence of sense of smell in producing, 183
  nature of, 188
  prophylaxis, 191
  proportion of, cases in the U. S. Navy, 180
  symptoms, 176
  synonyms, 175
  theories of, 181
    cerebral congestion, 187
    confusion of muscular sense, 185
    confusion of the sense of space, 186
    idiopathic indigestion, 186
    marine intoxication, 188
Seasickness, theories of, oscillations of cerebro-spinal fluid, 187
    shaking of brain, 186, 187
    shaking of abdominal viscera, 186
    spinal-cord origin, 187
    variations of barometric pressure, 185
    visual disturbances, 184, 186
  treatment, 189
  varieties of, 176
  vertigo in, 182
Sea-voyage, therapeutic uses of a, 195
Sewer's spasm, 483
Sewing-machine, effects of the, 486
Sheet-iron workers, deafness of, 451
Ship-painters, turpentine poisoning of, 402
Shock and Collapse, 141
  symptoms, 141; causes, 144; reaction, 155; prognosis, 157; pathology, 158; diagnosis, 162; prophylaxis, 163; treatment, 164; bibliography, 170
Shock, 141
  absence of distinctive lesions in, 162
  bibliography, 170
  causes, 144
  death from, 159
  definition, 141
  diagnosis, 162
    from cerebral concussion, 162
    from fat embolism, 163
    from syncope, 162
  distinction between collapse and, 144, 155
  erethistic, 141
  ether test in, 148
  from electricity, 151
  from poisoning, 155
  influence of anæsthesia upon, 147
    of hemorrhage upon, 150
    of the location of the injury in producing, 145
    of mental emotion in the production of, 151
    of the weather upon, 154
  insidious form of, 143
  irregular forms of, 143
  lessened susceptibility of women to, 151

Shock, lessening of, by will-power, 154
  local, 143
  medical, 155
  mental, 152
    secondary effects of, 153
  modified forms of, 143
  pathology, 158
  prognosis, 157
  prophylaxis, 163
  question of operating during, 147
  railroad accidents causing, 148
  reaction from, 155
    treatment in stage of, 170
  secondary effects of, 153
  symptoms, 141
  torpid, 141
  treatment, 164
  with excitement, 142
Shoddy fever, 427
Shoemaker, deformity of a, from machine working, 496
Shoemaker's spasm, 483
Shot-makers, diseases of, 344, 357
SHRADY, GEORGE F., on shock and collapse, 139
Sight, disturbances of, following heat-stroke, 284
Silver, poisoning by, 616
Silvering by amalgam, dangers of, 349
Singers, laryngeal neuroses of, 458
Sizing, injurious effects of, to the workmen, 424
Skin, affections of the, among workers in flax, 426
    among workers in quinine, 405
    among workmen in sugar refineries, 435
    in arsenical poisoning, 345
SMALL, BEAUMONT, on poisoning, 497
Smelters, diseases of, 343, 348, 356, 374, 379, 381, 389
Smoker's sore eye, 108
Smoking, hashish-, 88
  opium-, 72
  tobacco-, 106
Snuff, affections of workers in, 433
Soda, poisoning by, 582
Solanine, poisoning by, 554, see *Solanum*
Solanum carolinense, 554
  dulcamara, 554
  nigrum, 554
Solanum, poisoning by, 553
    symptoms, 554
    treatment, 555
Spine, railway, 149
STEWART, JAMES, on poisoning, 591
Stokers, effects of heat upon, 434
Stomach, acute alcoholic catarrh of the, 10
Stomatitis, mercurial, 352
Stone-cutters, diseases of, 420
Stramonium, poisoning by, 555
    symptoms, 556
    treatment, 557
Strychnine, poisoning by, 557
    fatal dose, 563
    illustrative cases, 563
    symptoms, 561
    treatment, 564
Strychnos nux vomica, 557
Sugar refineries, diseases of workmen in, 435
Sulphonal, poisoning by, 101
Sulphur, poisoning by, industrial, 386
Sulphuric acid, poisoning by, 568
    industrial, 388
    symptoms, 569
    treatment, 570
Sulphurous acid, poisoning by, industrial, 387,
Sunstroke, 253
  definition, 254; symptoms, 258; causation, 272; morbid anatomy, 275; prophylaxis, 276; treatment, 277; sequelæ, 283; bibliography, 285
Sunstroke and heatstroke, distinction between, made by certain writers, 261
  bibliography, 285
  cardiac form, 260
  causation, 272
  cerebro-spinal form, 259
  definition, 254
  forms of, 258, 259
  heart trouble following, 255, 283
  hyperpyrexia in, 259
  in children, 271
  insanity following, 284
  local, 272
  meningeal form, 259
  morbid anatomy, 572

Sunstroke, odor of, 259, 268
  pathology, 272
  prophylaxis, 276
  pulmonary apoplexy, 260
  sequelæ, 283
  statistics of, in the U. S. Army and Navy, 255
  symptoms, 258
  treatment, 277
  typical cases of, 263
  visual disturbances following, 284
Syncope, diagnosis of, from shock, 162
  heat, 258, see *Heat-stroke*

TEA, poisoning by, 112
    industrial, 406
    symptoms, 407
Tea-tasters, 406
Teeth, discoloration of, by copper, 377
Telegrapher's cramp, 473
  causes, 473
  illustrative cases, 478
  symptoms, 476
  treatment, 480
Telegraphic alphabet, 474
Temperature, body, at high altitudes, 205
    effects of temperature of surrounding atmosphere upon, 257, 292
Thebaine, 543
Theinism, 112, 406
  treatment, 114
Thorn apple, 555
Tin, diseases peculiar to workers in, 381
Tin-plate making, dangers of, 381
Tobacco, advantages of smoking, in certain cases, 110
  allowance of, in the treatment of alcoholism, 65
  antiseptic action of the smoke of, 110
  functional disorders caused by, 109
  poisoning by, 106
    chronic, 107
    industrial, 431
    treatment of, 107
Tobaccoism, 106
  and alcoholism, relations of, 111
  eye symptoms of, 108
Tobaccoism, symptoms, 107
  treatment of, 112
Tonsils, weavers', 425
Tool-grinding, injurious effects of, 414
Tremor, lead, 364, 599
  mercurial, 353, 613
Turpentine, poisoning by, industrial, 402
    symptoms, 402
    treatment, 403
Type-founders, lead-poisoning of, 357
Type-setters, diseases of, 357, 483

VENTILATION in workshops and factories, 452
Veratrine, 567
Veratrum, poisoning by, 566
    symptoms, 567
    treatment, 568
Vermilion, process of manufacture, 351
Vertigo in seasickness, 176, 182
Visual disturbances following heat-stroke, 284

WALL-PAPER makers, arsenical poisoning of, 343
Weavers, diseases of, 360, 424, 425, 427 453, 489
Women, employment of, in factories, 323
  lessened susceptibility of, to shock, 151
Wool dust, effects of, 427
Wool-sorters' disease, 428
  prevention, 430
  symptoms, 428
  treatment, 431
Wrist-drop, 363, 598
Writer's cramp, 462
  causes, 463
  diagnosis, 469
  morbid anatomy, 470
  pathology, 470
  prevention, 471
  prognosis, 470
  symptoms, 464
  treatment, 472

ZINC, poisoning by 611
    industrial, 379

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